TRANSACTIONS

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and
OTOLARYNGOLOGY

MARCH - APRIL
1961

Volume 65 **Number 2**

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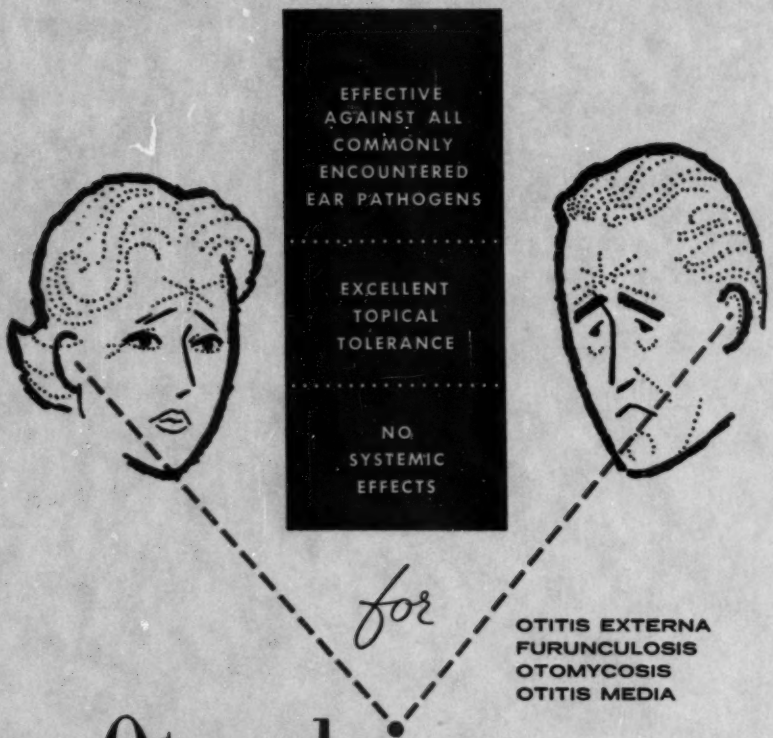

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TRANSACTIONS

American Academy of Ophthalmology and Otolaryngology

VOLUME 65 MARCH - APRIL, 1961 NUMBER 2

TABLE OF CONTENTS

PAGE

SYMPOSIUM: TONOGRAPHY

THE PRINCIPLE OF TONOGRAPHY: TERMS AND DEFINITIONS 133
PETER C. KRONFELD, M.D.

TONOGRAPHIC TECHNIQUE 136
ELMER J. BALLINTINE, M.D.

ERRORS IN TONOGRAPHY 145
ROBERT A. MOSES, M.D.

EXPERIMENTAL TONOGRAPHY 152
W. MORTON GRANT, M.D.

TONOGRAPHY IN THE DIAGNOSIS OF SIMPLE (OPEN ANGLE) GLAUCOMA. 156
BERNARD BECKER, M.D.

TONOGRAPHY IN THE MANAGEMENT OF GLAUCOMA 163
R. WINSTON ROBERTS, M.D.

ON APPLANATION TONOMETRY 171
THEODOR F. A. SCHMIDT, M.D.

BILATERAL CONGENITAL GLAUCOMA 178
MERRILL J. REEH, M.D.

BACTERIAL ALLERGY OF THE EYE 184
FREDERICK H. THEODORE, M.D.

A NEW TYPE SILICONE ROD FOR SCLERAL BUCKLING 197
A New Instrument
WILLIAM G. EVERETT, M.D.
MARGARET C. SHARRER, M.D.

(Continued on Next Page)

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TABLE OF CONTENTS *(Continued)*

PAGE

THE TRABECULOTOME 200
An Instrument for Trabeculotomy ab externo
LEE ALLEN
HERMANN M. BURIAN, M.D.

PROSTHETIC ARTICULATIONS IN STAPES SURGERY 202
VICTOR GOODHILL, M.D.

STREPTOMYCIN SULFATE AND DIHYDROSTREPTOMYCIN TOXICITY 222
T. MANFORD MCGEE, M.D.

A SURGICAL OTOSCOPE 230
A New Instrument
BEN H. SENTURIA, M.D.

A MIDDLE EAR INTUBATOR 233
A New Instrument
ALBERT H. WILLIAMS, M.D.

EDITORIALS

COMMITTEE ON CONSERVATION OF HEARING SUBCOMMITTEE FOR LISTING OF AUDIOMETERS: LISTING PROCEDURE AND AGREEMENTS 235

ACADEMY PLANNED TOUR 236

LETTERS TO THE EDITOR 238

HOME STUDY COURSES 237

IN MEMORIAM 239

LISTING OF AUDIOMETERS 240

NEWS AND ANNOUNCEMENTS XXXI

POSITIONS XXXVII

DIRECTORY OF OPHTHALMIC AND OTOLARYNGIC SOCIETIES XXXIX

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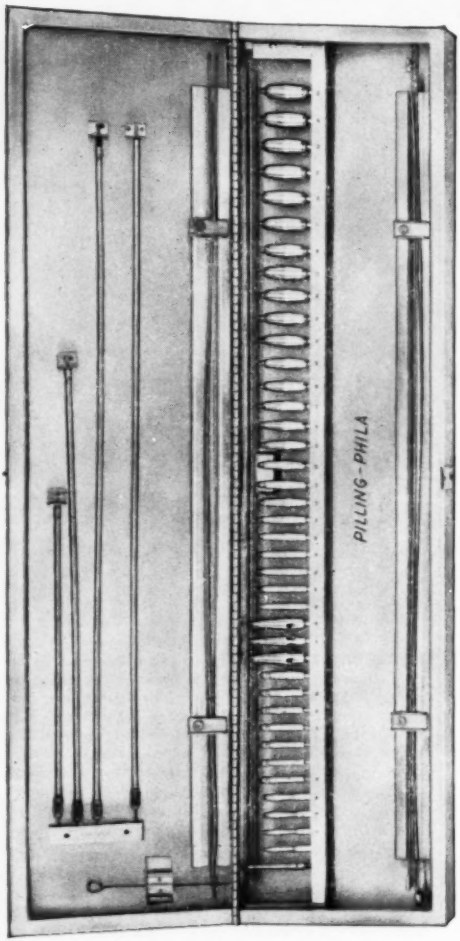

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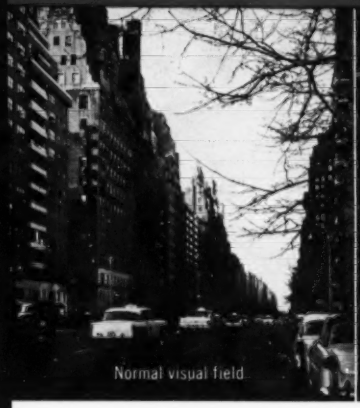
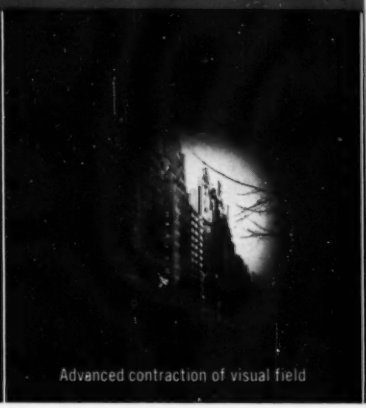


Normal visual field

Beginning contraction of visual field



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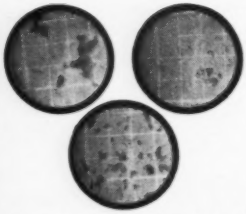
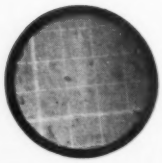
THERE ARE DIFFERENCES IN STEROID PREPARATIONS FOR THE EYE

IN THE SUSPENSION

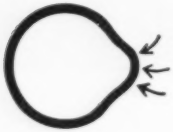


Appearance of typical ophthalmic steroid preparations (400 x).

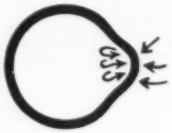


98% of Alcon's prednisolone particles measure less than 5 μ (400 x).

IN THE PENETRATION

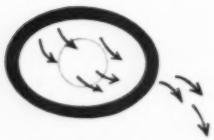


Water soluble steroids provide surface action only.

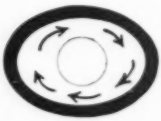


Alcon's form of prednisolone provides surface action *plus* corneal penetration.

IN THE VEHICLE



Simple aqueous vehicles are quickly washed from the eye. Contact time is 1-2 minutes.



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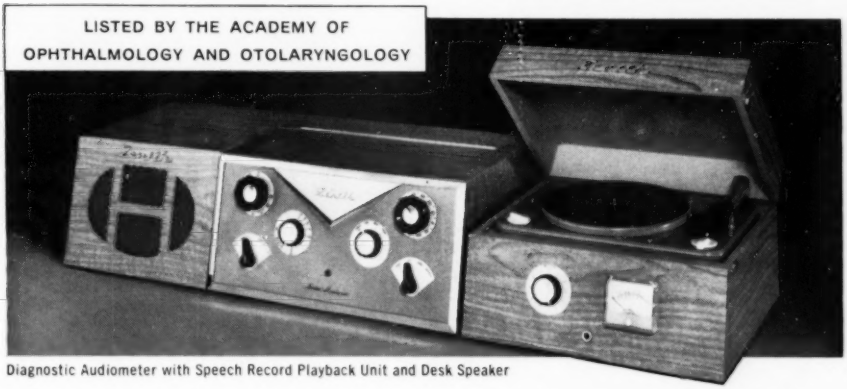

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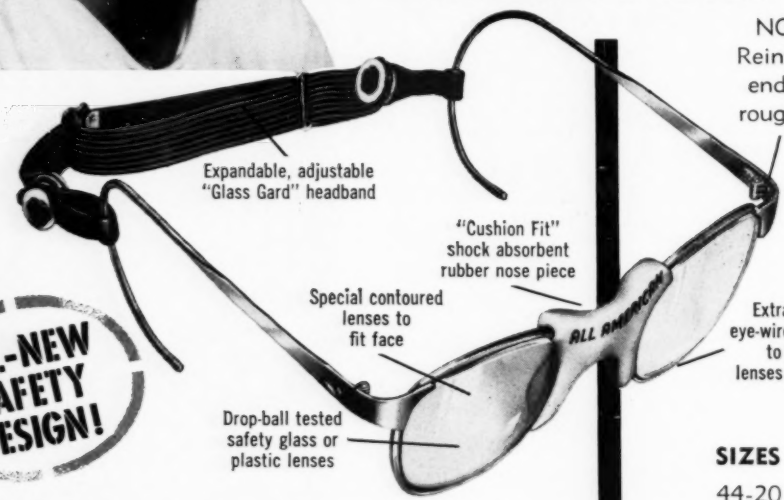
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1. *Am. J. Digest. Dis.* 22:5, 1955.
2. *Southeastern Med.* 40:120, 1959.
3. *Am. J. Ophth.* 42:771, 1956.
4. *M. Times* 84:741, 1956.

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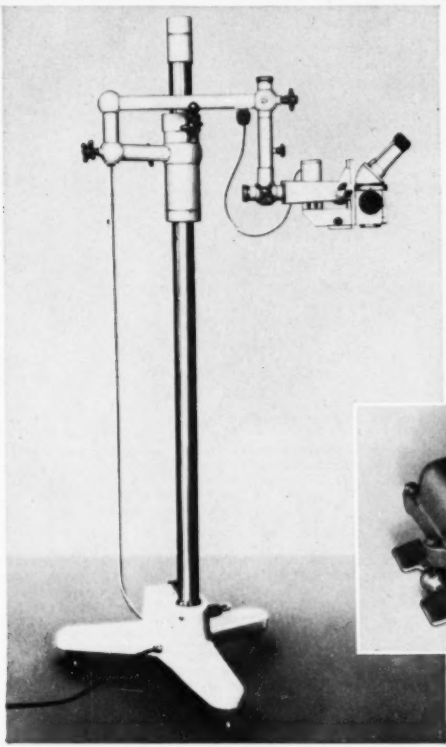

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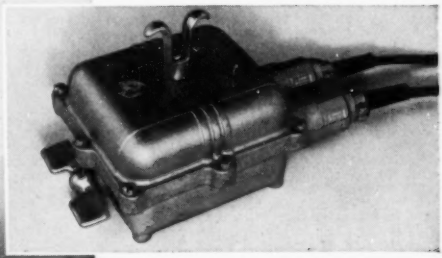

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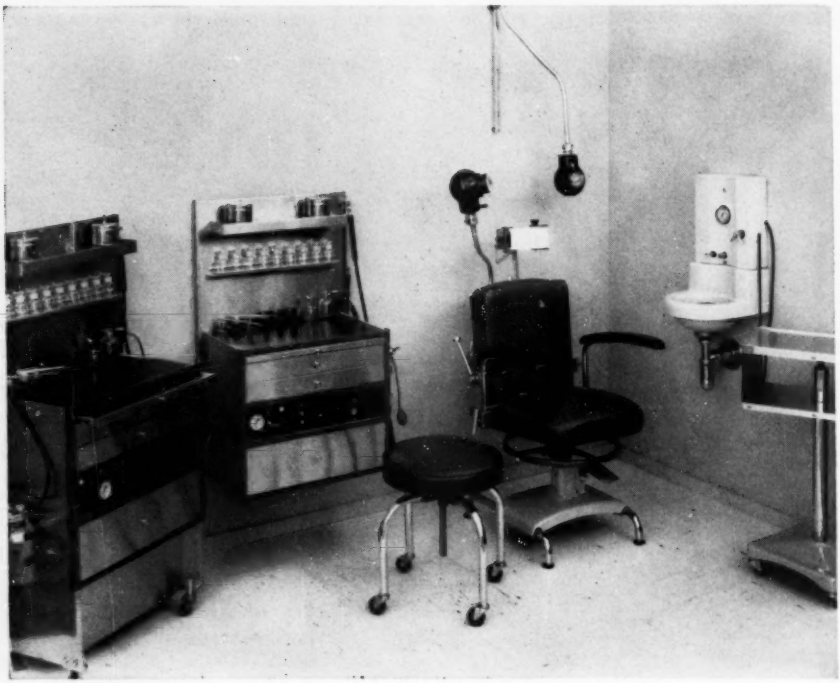

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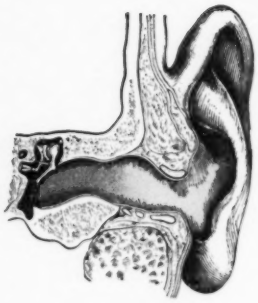

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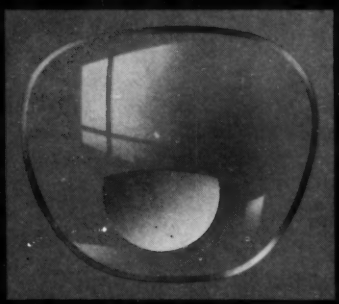

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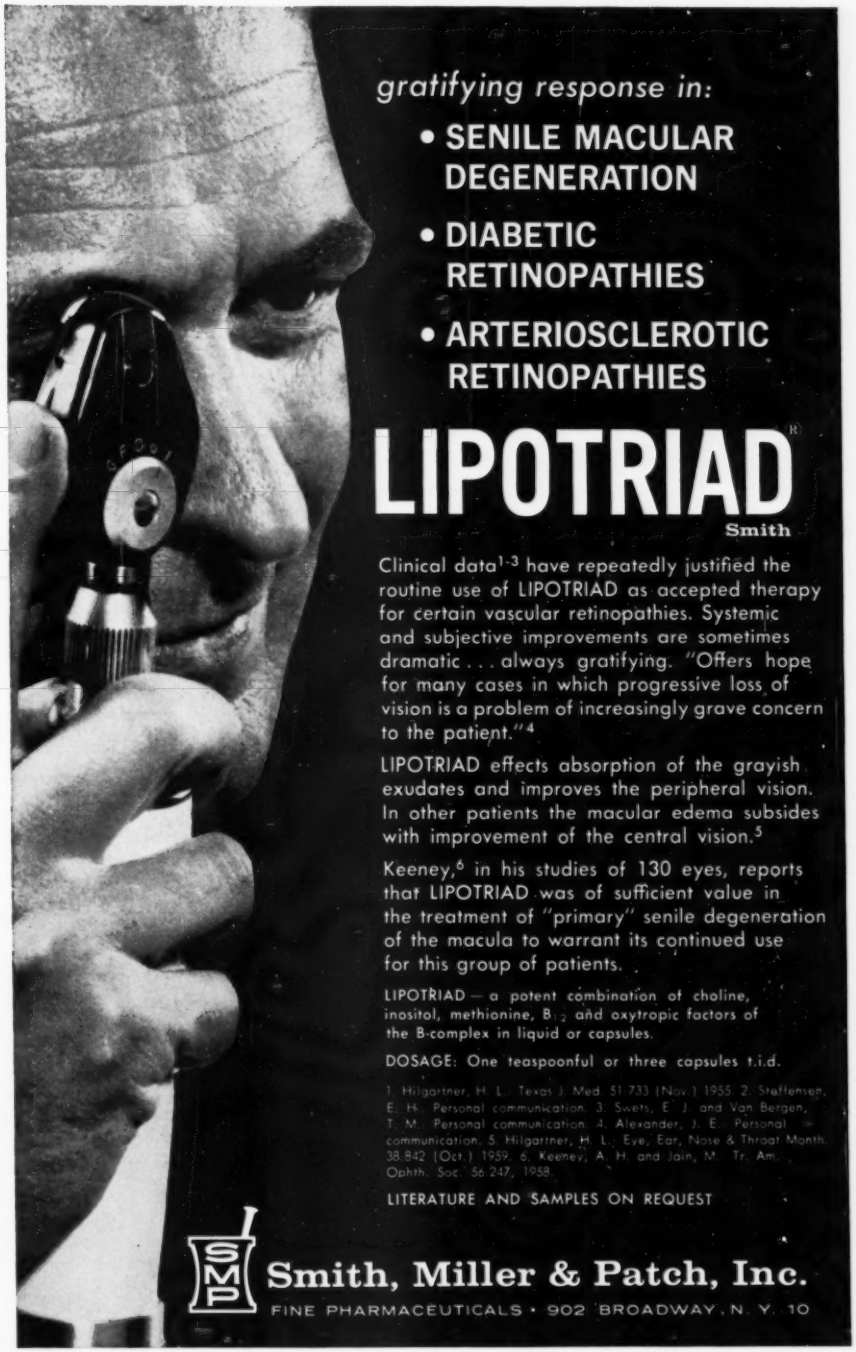

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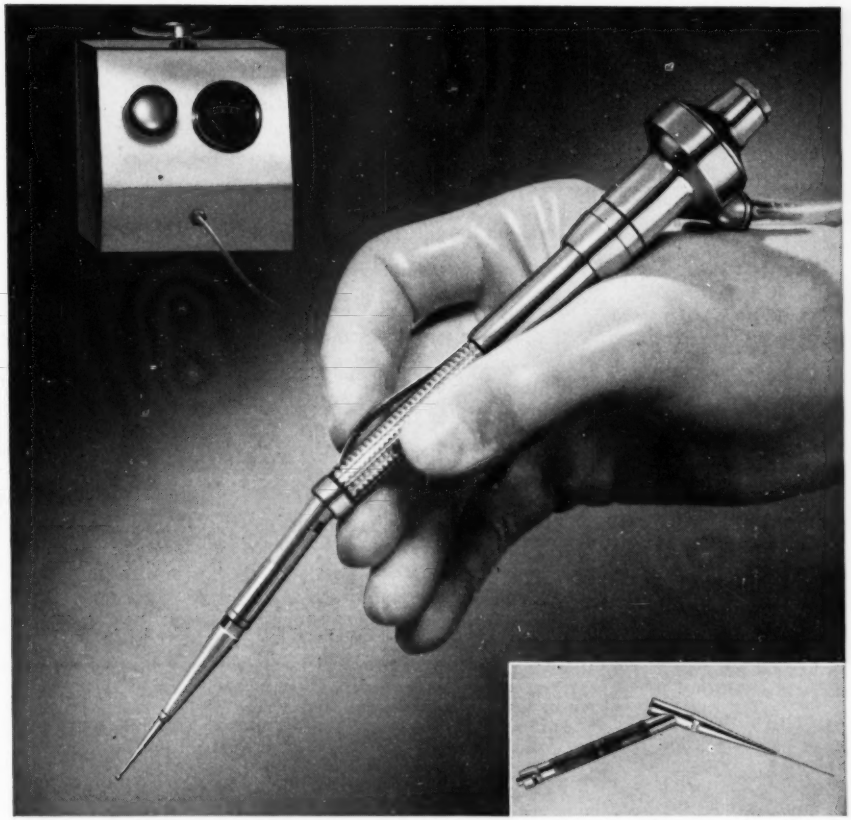

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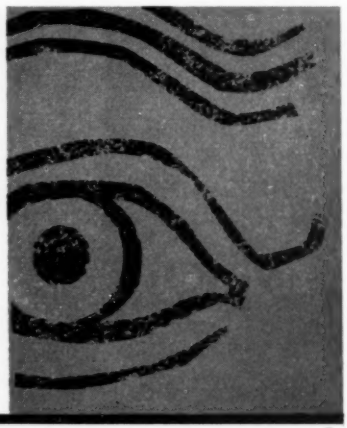

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Symposium: Tonography

THE PRINCIPLE OF TONOGRAPHY: TERMS AND DEFINITIONS

PETER C. KRONFELD, M.D.
CHICAGO, ILLINOIS

GENERALLY and literally the term *tonography* means the observation and the recording of pressure changes in any physical or biological system. Grant,[6] in 1950, applied the term *tonography* to the recording of the changes in intraocular pressure which occur during and as the result of prolonged tonometry with an instrument of the Schiøtz type. A steady increase of the tonometric reading (or decrease of the intraocular pressure) had been noted under these circumstances by earlier investigators and has been interpreted as a result of expulsion of intraocular fluid by the excess pressure created by the application of the tonometer. For the magnitudes of these two factors Friedenwald's calibration studies provided estimates which were used by Grant to express the change in tonometric scale readings during prolonged tonometry in terms of units of intraocular fluid expelled, divided by units of pressure increment and by time, that is:

a ratio designated by Grant as the "coefficient of the facility of aqueous outflow (C)."

$$\frac{\Delta V}{\text{average } \Delta P \times \text{time}},$$

The interpretation of the tonogram in terms of ease of aqueous expulsion is the principle of tonography. Implicit in this interpretation is the assumption that during the test period the expulsion of aqueous dominates the changes of the other factors possibly involved, that is: (1) the intraocular blood volume; (2) the rate of aqueous flow; (3) the distensibility of the ocular coats, and (4) the plastic properties of the cornea.

Once an estimate for the facility of outflow or of its reciprocal, the resistance to outflow, was available, its use for the calculation of the rate of aqueous flow under steady-state conditions was the logical next step. Applied to the outflow channels of the eye, the general flow formula,

$$\text{flow} = \frac{\text{pressure gradient}}{\text{resistance}},$$

may be written as follows:

$$\text{flow} = (\text{intraocular pressure} - \text{episcleral venous pressure}) \times \text{facility of outflow}.$$

Estimates for the intraocular pressure, the episcleral venous pressure and the facility of outflow are obtained by measurement. From these measurements the rate of aqueous flow is calculated.

Grant's work had a profound effect on ophthalmology. The new method, in its original or modified form, was applied to normal and diseased eyes, un-

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der steady-state as well as under experimentally or pharmacologically altered conditions. The results obtained in glaucomatous eyes by many different investigators were remarkably uniform. The test-retest consistency of a good many of the findings and the high level of agreement between tonography, on the one hand, and perfusion studies and flow rate measurements by other methods, on the other, caused a number of observers to adopt and recommend tonography as a routine method for the study of aqueous dynamics in the living, normal or diseased, eye.[1,2,7] Within limits of which these workers are very much aware, they have considered tonography a valid and valuable method for the estimation of the capacity or competence of the outflow channels, and of the rate of aqueous flow.

In open disagreement with these workers are several other groups of investigators[3,5,10] who are appalled by the number of assumptions and by the magnitude of the difficulties and irregularities encountered in initiating tonography; these investigators are understandably hesitant to base any physiological concepts or clinical decisions upon tonographic data.

An objective investigation into the validity of tonographic data seemed in order. A Subcommittee of the Committee on Standardization of Tonometers of the Academy selected a panel of men who had been actively engaged in tonography since its very beginning. In preparation for their presentation today these men have reanalyzed and reviewed their own data as well as those of other workers in this field. The data upon which they base their opinions and the results of this review are presented herewith.

Symbols and Terms

The symbols and terms used in tonography may require explanation. The symbol most easily defined is P_t, the *intraocular pressure during tonometry*, that is, while the tonometer is resting on the eye. P_t values corresponding to the *scale readings* (R) of standardized Schiøtz tonometers have been determined experimentally on enucleated human eyes by a number of investigators. The results of these measurements have varied considerably, due to factors which are gradually becoming better understood, such as the method of cannulation, the mode of support of the eyeball during the test, and the condition of the eye.

P_o is the *starting point of tonography*, the intraocular pressure prevailing just before the application of the tonometer or "steady-state" pressure, which we aim to determine by tonometry. The P_o values used in this symposium are those of the conversion scale of 1955.

Since at the very beginning of tonography, particularly in patients unfamiliar with the test, a number of minor disturbances may occur in the tonogram, the real initial reading may have to be determined by extrapolating the curve to zero time.

The change in intraocular fluid contents during tonography ($\triangle V$) consists of several parts: the deformation and indentation of the cornea by footplate and plunger of the tonometer, the distension of the entire eyeball wall by the tonometric rise in pressure and, possibly, a distinct indentation of the posterior segment. These components have been measured in human eyes, again with somewhat discordant results.

Of very considerable significance in tonography is the ocular rigidity which is a mathematical expression of the empirical relationship between volume and pressure in the eyeball. The analysis of measurements by earlier investigators led Friedenwald[4] to the concept that ocular rigidity is independent of the absolute pressure. Very recently this

concept has been confirmed by *in vivo* and *in situ* measurements on human eyes.[9] The *coefficient of ocular rigidity* (K or E) is the slope of the graph that relates the logarithm of the pressure change to the volume change.

The symbol P_v applies to the *average pressure* in the small episcleral veins or their aqueous vein feeders near the limbus. Measurements of this pressure by investigators with special experience and skill in this field have yielded values in the neighborhood of 10 mm. of mercury. This value has been used for all calculations of the rate of aqueous flow quoted in this symposium. During tonography this pressure rises slightly and thereby reduces the excess pressure created by the application of the tonometer. An appropriate correction factor has been determined by Linnér[8] and applied to all C values quoted in this symposium.

The *rate of aqueous flow* (F) is estimated by calculation and expressed in cubic millimeters or microliters per minute.

For all tonographic factors, the panel has used Friedenwald's 1955 estimates, because they have been most widely used for the evaluation of tonographic data and yield results which are consistent with themselves and with measurements by other methods.

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TONOGRAPHIC TECHNIQUE

ELMER J. BALLINTINE, M.D.
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PRACTICALLY, tonography consists in making a record of the readings of a Schiøtz tonometer while the tonometer rests on the eye for four minutes or longer. While gross statistical correlations between various aspects of glaucoma and tonographic results can be made from records obtained by applying a nonrecording mechanical or electrical tonometer to an eye for four minutes, all the members of the panel agree that in order to obtain records of useful precision in individual cases, an electric tonometer and some form of automatic recording must be used. Dr. Roberts has published results of an extensive study of this question, and they indicate the large uncertainties that are introduced by attempting to perform tonography without automatic recording.

For example, in a series of 539 tonograms from patients having simple glaucoma, the tonograms were obtained with a recorder; simultaneously the tonometer readings at half-minute intervals were written down. The value of C was then calculated from the two sets of data. The value found by automatic recording was considered to be the true value. The standard deviation of C calculated from the half minute values was .04. This means that for a tonogram obtained without automatic recording, deviations of .08 or greater from the value of C obtainable with automatic recording can

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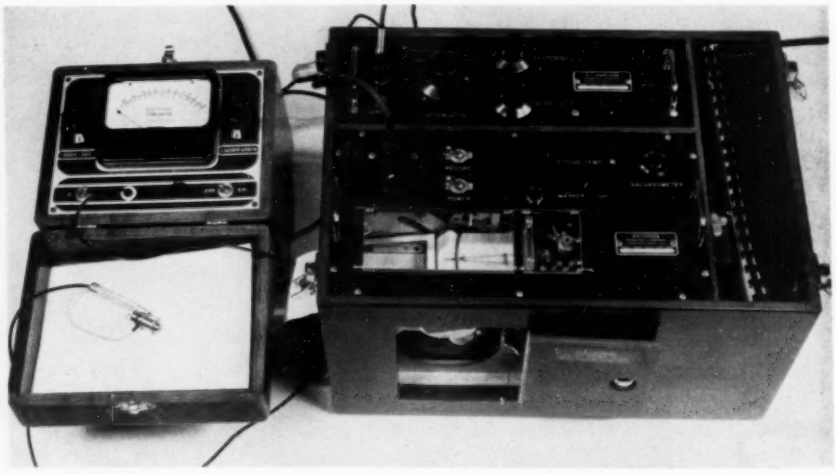

FIG. 1—Apparatus for tonography. Electric tonometer (V. Mueller and Company) driving a direct current amplifier and recording millivolt meter (Sanborn Company).

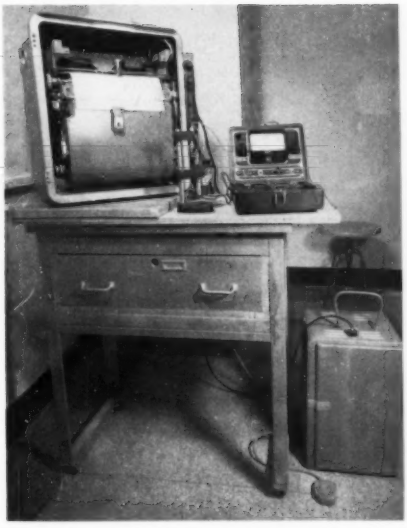

FIG. 2—Apparatus for tonography. Electric tonometer driving a recording potentiometer (Leeds & Northrup).

be expected in 5 per cent of the cases. Deviation of .04 or more can be expected in 30 per cent of tonograms made without automatic recording.

There are presently available three systems for making the tonographic record. In all systems the Mueller electric tonometer produces an electrical signal proportional to the tonometer readings. This signal is recorded either (1) by a recording millivolt meter driven by a direct current amplified (fig. 1); (2) by a recording potentiometer (fig. 2), or (3) by a recording milliammeter (fig. 3). Of these systems, the DC amplifier-millivolt meter recorder is probably most sensitive to instantaneous minute variations in the output signal, but the value of such rapid response is problematic in view of the large damping of such small variations introduced by the filtering cir-

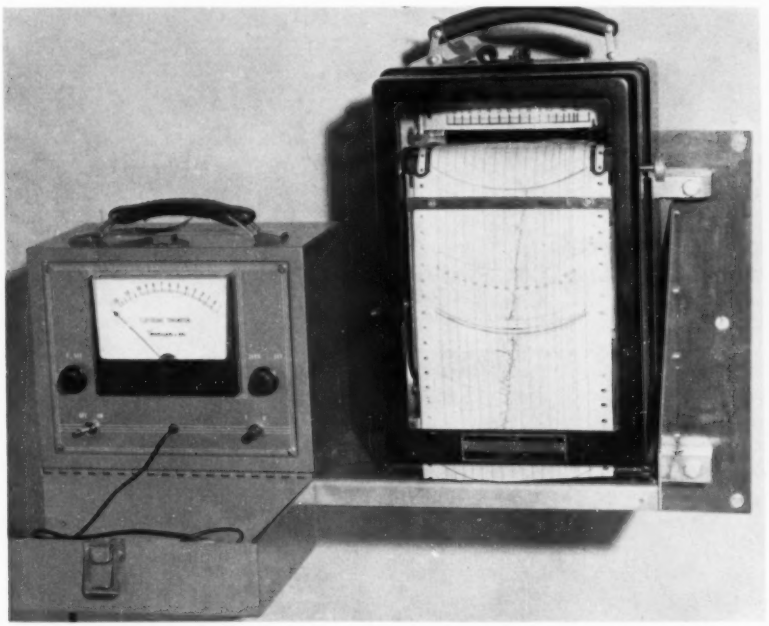

FIG. 3—Apparatus for tonography. Electric tonometer driving a recording milliammeter (Esterline Angus).

cuits in the tonometer itself. The potentiometer instrument has the advantage of a wide scale so that fractional tonometric scale divisions are easily read. Although the direct recording milliammeter is less sensitive to instantaneous variations in the output signal from the tonometer and tends to distort such variations because of the electrical and mechanical inertia of its pen motor, it has the great advantages of simplicity, wide scale, availability and relatively low cost. In my experience the slight distortions mentioned are of no consequence.

The calibration is printed on the charts of some instruments; with others, it is necessary to record the calibration scale on each tonogram. The beginning of the tonogram ("zero time") and the point corresponding to 4 minutes should be marked. A smooth curve averaging the mid-points of the tonometer fluctuations should be drawn through the record. Some technicians insist on drawing

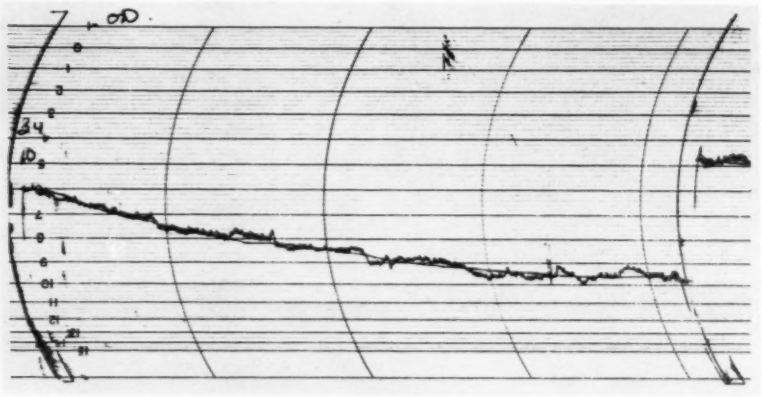

FIG. 4—A normal tonogram.

A normal tonogram made with the recording milliammeter (Esterline Angus Recorder) is shown in figure 4. The tracing shows the ocular pulse, slight variations in intraocular pressure synchronous with respiration, and variations which are synchronous with the Traube-Hering waves of blood pressure variation. If the tonometer has so much friction between plunger and footplate that the ocular pulse is not well demonstrated, it is unlikely that the record will be useful.

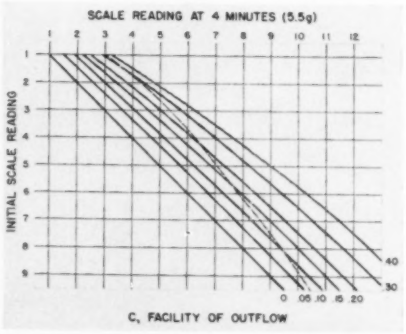

FIG. 5—Nomograph for finding C for tonograms made with the 5.5 gm. tonometer weight. The dotted line is the locus of paired tonometer readings for $P_o/C = 100$. Points falling to the left of the line have values of $P_o/C > 100$. Points lying to the right of the line have values of $P_o/C < 100$. (From Grant, recalculated by Ballintine.)

TABLE I

TABLE FOR FINDING THE CO-EFFICIENT OF OUTFLOW (C) FOR TONOGRAMS MADE WITH THE 5.5 GRAM WEIGHT (ADAPTED FROM BECKER)

INITIAL READING		5.5 gm. wt. ΔR (CHANGE IN SCALE READING)										
Po	R	0	.50	1.00	1.50	2.00	2.50	3.00	3.50	4.00	4.50	5.00
24	3.00	0	.04	.09	.14	.20	.27	.35	.44	.54	.65	.78
23	3.25	0	.04	.09	.14	.20	.26	.33	.42	.51	.62	.74
22	3.50	0	.04	.08	.13	.19	.25	.32	.40	.49	.59	.70
22	3.75	0	.04	.08	.13	.19	.25	.31	.38	.47	.56	.66
21	4.00	0	.04	.08	.13	.18	.24	.30	.37	.45	.54	.63
20	4.25	0	.04	.08	.13	.18	.24	.30	.36	.43	.52	.60
19	4.50	0	.04	.08	.12	.17	.23	.29	.35	.42	.50	.58
18	4.75	0	.04	.08	.12	.17	.23	.28	.34	.41	.48	.56
17	5.00	0	.04	.08	.12	.17	.22	.27	.33	.40	.47	.54
17	5.25	0	.04	.08	.12	.17	.22	.27	.33	.39	.46	.53
16	5.50	0	.04	.08	.12	.16	.21	.26	.32	.38	.45	.52
15	5.75	0	.04	.08	.12	.16	.21	.26	.32	.38	.44	.50
15	6.00	0	.03	.07	.11	.15	.20	.25	.31	.37	.43	.49
14	6.25	0	.03	.07	.11	.15	.20	.25	.31	.37	.43	.49
13	6.50	0	.03	.07	.11	.15	.20	.25	.30	.36	.42	.48
13	6.75	0	.03	.07	.11	.15	.20	.24	.30	.36	.41	.47
12	7.00	0	.03	.07	.11	.15	.20	.24	.29	.35	.40	.46
11	7.50	0	.03	.07	.11	.15	.19	.24	.29	.34	.39	.45
10	8.00	0	.03	.07	.11	.15	.19	.24	.29	.34	.39	.45
9	8.50	0	.03	.07	.11	.15	.19	.23	.28	.33	.39	
9	9.00	0	.03	.07	.11	.15	.19	.23	.28	.33		
8	9.50	0	.03	.07	.11	.15	.19	.23	.28			
7	10.00	0	.03	.07	.11	.15	.19	.23				
6	11.00	0	.03	.07	.11	.15						

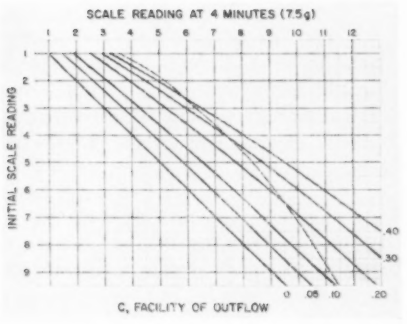

FIG. 6—Nomograph for finding C for tonograms made with the 7.5 gm. tonometer weight. The dotted line has the same significance as described for figure 5. (From Grant, recalculated by Ballintine.)

a straight line through the curve with a ruler. There is no justification for this practice, which can only result in loss of useful information. The scale readings at 0 time and at 4 minutes should then be read from the curve drawn. They should not be read directly from the tonographic record. The readings should be estimated to the nearest quarter scale division. When the initial and 4-minute readings are known, the value of the intraocular pressure corresponding to the initial scale reading is read from the conventional Schiøtz calibration table. The intraocular pressure should be recorded only to the nearest millimeter of

TABLE II

TABLE FOR FINDING THE CO-EFFICIENT OF OUTFLOW (C) FOR TONOGRAMS MADE WITH THE 7.5 GRAM WEIGHT (ADAPTED FROM BECKER)

INITIAL READING		7.5 gm. wt. ΔR (CHANGE IN SCALE READING)										
Po	R	0	.50	1.00	1.50	2.00	2.50	3.00	3.50	4.00	4.50	5.00
36	3.00	0	.03	.07	.12	.17	.23	.31	.40	.50	.62	.76
34	3.25	0	.03	.07	.12	.17	.22	.29	.37	.46	.57	.69
33	3.50	0	.03	.07	.11	.16	.21	.28	.35	.43	.53	.63
32	3.75	0	.03	.07	.11	.16	.21	.26	.33	.41	.49	.59
30	4.00	0	.03	.06	.10	.15	.20	.25	.32	.39	.46	.55
29	4.25	0	.03	.06	.10	.15	.19	.25	.30	.37	.44	.52
28	4.50	0	.03	.06	.10	.14	.18	.24	.29	.35	.42	.50
27	4.75	0	.03	.06	.10	.14	.18	.23	.28	.34	.40	.47
26	5.00	0	.03	.06	.10	.13	.17	.22	.27	.33	.39	.45
25	5.25	0	.03	.06	.10	.13	.17	.22	.27	.32	.38	.43
24	5.50	0	.03	.06	.09	.13	.16	.21	.26	.31	.37	.42
23	5.75	0	.03	.06	.09	.13	.16	.21	.26	.31	.36	.41
22	6.00	0	.03	.06	.09	.12	.16	.20	.25	.30	.35	.40
21	6.25	0	.03	.06	.09	.12	.16	.20	.25	.29	.34	.39
20	6.50	0	.03	.05	.09	.12	.15	.19	.24	.28	.33	.38
19	6.75	0	.03	.05	.09	.12	.15	.19	.24	.28	.33	.38
18	7.00	0	.03	.05	.08	.12	.15	.19	.23	.27	.32	.37
17	7.50	0	.03	.05	.08	.12	.15	.19	.23	.27	.31	.36
16	8.00	0	.03	.05	.08	.11	.15	.18	.22	.26	.30	.35
14	8.50	0	.03	.05	.08	.11	.15	.18	.22	.26	.30	
13	9.00	0	.03	.05	.08	.11	.15	.18	.22	.25		
12	9.50	0	.03	.05	.08	.11	.15	.18	.22			
11	10.00	0	.03	.05	.08	.11	.14	.18				
10	11.00	0	.03	.05	.08	.11	.14					

mercury. The recording of decimal fractions for intraocular pressure implies a precision that does not exist.

The value of C is found either by using the nomograph (figs. 5 and 6) or from tables I, II and III. To use the nomograph, find the scale reading at zero time on the vertical axis at the left of the chart. Trace the horizontal line through this point to the right until the vertical line passing through the point on the upper horizontal axis corresponding to the 4-minute reading is reached. The point of intersection will be on or close to one of the diagonal lines that are the locus of the pairs of readings corresponding to the value of C marked at the lower right end of each line. If the point lies between two diagonal lines, the value of C is found by interpolating between the values marked on the two lines.

If the tables are used, the initial scale reading (R_o) is found in the left-hand column; this row is then traced to the left until the column headed Δ R, equal to the difference between the four minute reading (R_4) and R_o is reached. The figure in this row and column is C for the tonogram being examined.

TABLE III

TABLE FOR FINDING THE CO-EFFICIENT OF OUTFLOW (C) FOR TONOGRAMS MADE WITH THE 10.0 GRAM WEIGHT (ADAPTED FROM BECKER)

INITIAL READING		10 gm. wt. △R (CHANGE IN SCALE READING)										
Po	R	0	.50	1.00	1.50	2.00	2.50	3.00	3.50	4.00	4.50	5.00
51	3.00	0	.03	.06	.10	.15	.21	.29	.38	.49		
49	3.25	0	.03	.06	.10	.14	.20	.27	.35	.44		
47	3.50	0	.03	.06	.09	.13	.19	.25	.32	.40	.50	
45	3.75	0	.03	.06	.09	.13	.18	.23	.30	.37	.46	
43	4.00	0	.02	.05	.08	.12	.17	.22	.28	.35	.43	.52
42	4.25	0	.02	.05	.08	.12	.17	.21	.26	.33	.40	.48
40	4.50	0	.02	.05	.07	.11	.16	.20	.25	.31	.38	.44
38	4.75	0	.02	.05	.07	.11	.16	.20	.24	.29	.36	.42
37	5.00	0	.02	.05	.07	.10	.15	.19	.23	.28	.34	.40
36	5.25	0	.02	.05	.07	.10	.15	.19	.23	.27	.32	.38
34	5.50	0	.02	.05	.07	.10	.14	.18	.22	.26	.31	.36
33	5.75	0	.02	.05	.07	.10	.14	.18	.22	.25	.30	.34
32	6.00	0	.02	.04	.07	.10	.13	.17	.21	.24	.29	.33
31	6.25	0	.02	.04	.07	.10	.13	.17	.21	.24	.28	.32
29	6.50	0	.02	.04	.07	.10	.13	.16	.20	.23	.27	.31
28	6.75	0	.02	.04	.07	.10	.13	.16	.20	.23	.27	.31
27	7.00	0	.02	.04	.07	.09	.12	.15	.19	.22	.26	.30
26	7.25	0	.02	.04	.07	.09	.12	.15	.19	.22	.26	.30
25	7.50	0	.02	.04	.07	.09	.12	.15	.18	.21	.25	.29
24	7.75	0	.02	.04	.07	.09	.12	.15	.18	.21	.25	.29
23	8.00	0	.02	.04	.06	.09	.12	.14	.18	.21	.24	.28
21	8.50	0	.02	.04	.06	.09	.11	.14	.18	.20	.23	.27
20	9.00	0	.02	.04	.06	.09	.11	.14	.17	.20	.23	.26
18	9.50	0	.02	.04	.06	.09	.11	.14	.17	.20	.23	.26
16	10.00	0	.02	.04	.06	.09	.11	.14	.17	.19	.22	.26
14	11.00	0	.02	.04	.06	.09	.11	.14	.17	.19	.22	.25

The tonometric and tonographic charts and tables are valid only for initial readings of 3.5 or larger. If the initial reading (R_o) is less than 3.5, the next heavier tonometer weight should be used while recording the tonogram.

A preliminary measurement of the intraocular pressure with the applanation tonometer will indicate which weight to use for tonography and alert the examiner to the possibility of abnormal ocular rigidity if the intraocular pressure indicated by the Schiøtz tonometer is different from the applanation reading. If the applanation reading is greater than 22, the 7.5 gm. weight should be used for tonography.

The average value of C found by Becker in 909 presumably normal eyes was .28 with a standard deviation of 0.05.

The members of the panel have found that the consensual fall in intraocular pressure of about 2 mm. Hg in the second eye which occurs frequently while

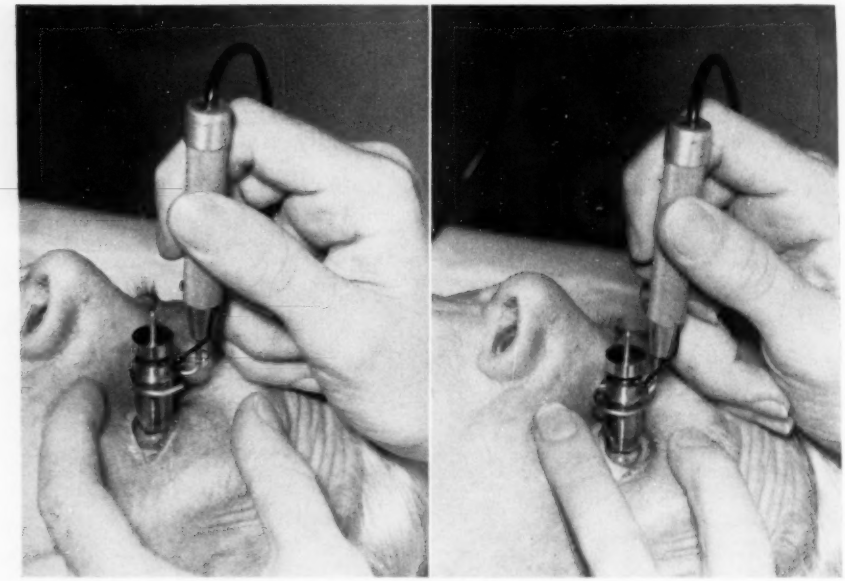

FIG. 7—(Left) Correct method of retracting lids during tonography. (Right) Incorrect method of retracting lids. This method almost invariably results in directing some finger pressure into the orbit and against the globe.

tonography is being performed on the first eye does not affect the interpretation of the tonogram obtained a few minutes later from the second eye.

It is the experience of the members of the panel that errors made by the operator are the commonest reason for obtaining unsatisfactory tonographic records. It should be obvious that in retracting the lids no pressure should be directed into the orbits (fig. 7), that the patient should be reassured and made comfortable, that the instrument should be held vertically on the cornea, and that the patient's fixation should be controlled so that the axis of the cornea is vertical. Nevertheless, in attempting to help others establish tonographic laboratories, obvious errors in these procedures are

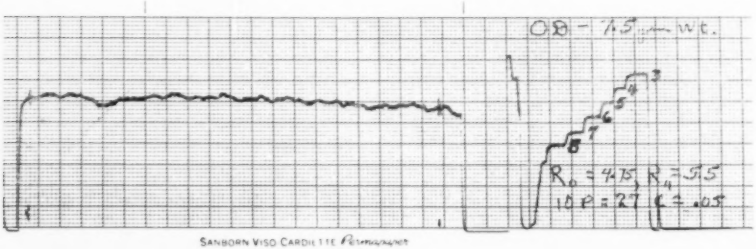

FIG. 8—Tonogram, defective because the tonometer scale is compressed on the record.

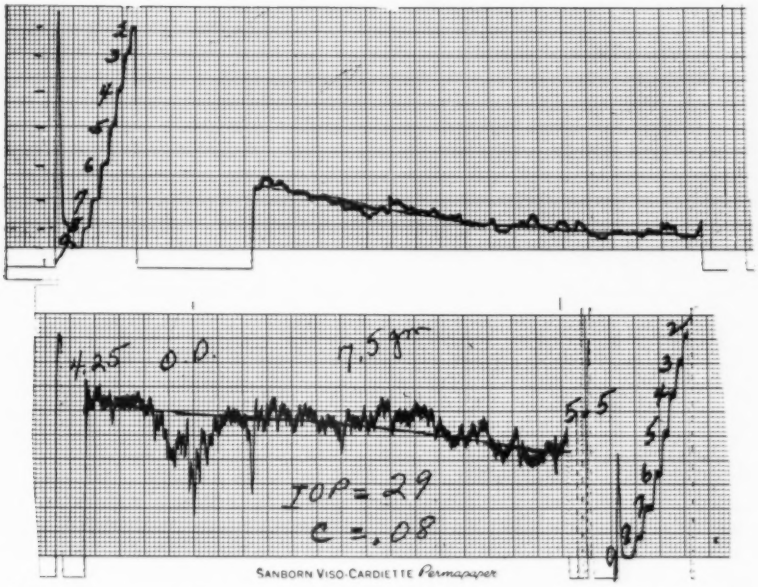

FIG. 9—Upper record: Tonogram made with a tonometer with excessive damping. Lower record: Tonogram from opposite eye of same patient made with tonometer having satisfactory damping.

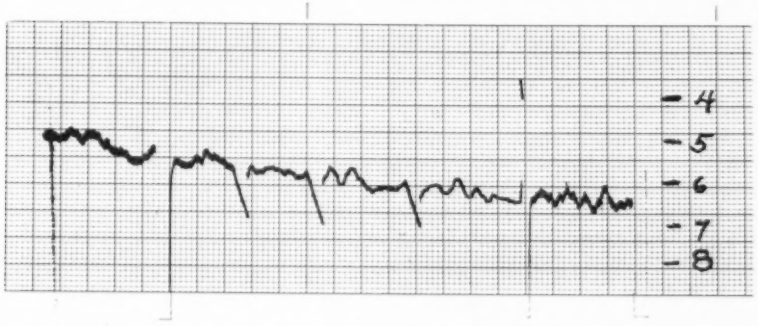

FIG. 10—Effect of adding increments of damping to the filter circuit in the tonometer output during tonography. In the last portion of the record, the excessive filter capacity has been removed.

met repeatedly. Occasionally, technicians will be encountered who continue to turn out inferior tonograms despite repeated instruction. The only solution is to find a new technician for tonography.

It is unfortunate that there are electric tonometers in the hands of ophthalmologists today which are not suitable for tonography because of defects in their electrical performance. These defects are of two main types. First, the electrical output of the tonometer may be so low that, with the available recorders, the scale divisions as recorded

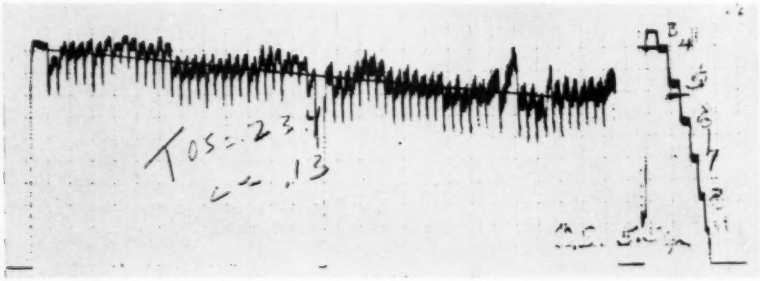

FIG. 11—Tonogram showing the momentary fall in intraocular pressure that occurs each time there is a dropped ventricular systole during partial heart block.

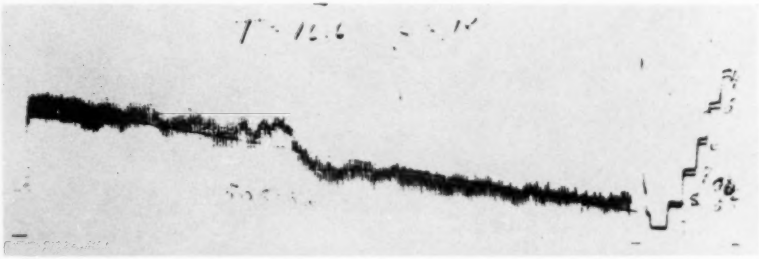

FIG. 12—Tonogram showing the effect of mild steady pressure applied inadvertently during the first part of the record. At the discontinuity near the middle of the record, the technician discovered her error and for the rest of the tonogram the lids were retracted properly.

on the chart are so narrow that precise readings are not possible (fig. 8). For clinical tonography, the combined tonometer-recording system must produce a record on which one tonometer scale division covers at least 7 mm. of the chart width. If the tonometer does not have an output to produce scale indications of this size, it is necessary to return it to the manufacturer with an explanation of the defect so that the instrument can be readjusted to produce an adequate output.

The second defect commonly encountered is that the output signal of the electric tonometer is so highly damped by the filtering circuits in the tonometer that the recording instrument performs sluggishly. When the tonometer is placed on the eye, the true tonometer reading is not recorded until after several seconds have elapsed (fig. 9 and 10). This sluggishness of response can be remedied only by replacing the filtering circuits on the tonometer output. It is futile to attempt to perform clinical tonography with instruments having either of these defects.

With experience it is possible to judge the nature of irregularities present in the tonographic records. For example, irregularities of the cardiac rhythm, especially dropped beats, can be diagnosed from the variations in the ocular pulse (fig. 11). Excess lid pressure from squeezing or improperly held lids occasionally produces characteristic variations in the tonogram (fig. 12). Excess friction between plunger and footplate also produces characteristic changes. All such records must be considered unreliable and less weight given to their interpretation than when the record shows evidence of good technique.

ERRORS IN TONOGRAPHY

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I PROPOSE to discuss only the most frequent sources of difficulty in interpretation of tonograms. These fall into the following categories:

1. Nonsteady state of the eye.
2. Ocular rigidity corrections.
3. Rapid drop of pressure in the first minute.
4. Operator errors.
5. Patient errors.
6. Errors deriving from the tonometer itself.

Some of these factors have already been alluded to by Dr. Kronfeld and Dr. Ballintine.

NONSTEADY STATE OF THE EYE

The theory of tonography presupposes that before tonography is begun, inflow and outflow of aqueous are equal, that facility of outflow is constant, and that vascular pressures are stabilized. In short, the theory demands that the eye be in a state of equilibrium. Application of the tonometer produces an increase in intraocular pressure and an imbalance in the equilibrium which is measurable provided that production of aqueous, facility of outflow and vascular pressures are not altered in an unknown way. Linnér has estimated that episcleral venous pressure is raised about 1.25 mm. Hg by the application of the tonometer, and this change in the equilibrium can be incorporated in the tonographic formula.

I will illustrate with an example of tonography in an eye in which equilibrium before tonography does not exist. Let us say that open angle glaucoma has been diagnosed in a patient. The intraocular pressure is 43 mm. Hg. The examiner places a drop of pilocarpine in the patient's eye, and then orders a tonogram. Some minutes later the tonogram shows a P_o of 35, and a very good coefficient of facility of outflow, 0.40 (fig. 1). What can this mean—an elevated pressure and excellent facility? The tonogram means little because the eye was not in a steady state. The pilocarpine had improved the facility of outflow, and pressure was dropping toward a new equilibrium level, but it hadn't reached that level yet and so there was no true equilibrium P_o to enter in the formula for calculation of C.

A second example of nonequilibrium might be seen in a patient with very narrow angles who is given a darkroom test. During the darkroom test the patient's iris occludes his angle and the intraocular pressure rises. Application of the tonometer may displace enough aqueous to force the iris away from the trabecular area, again producing the paradoxical finding of elevation of pressure and an apparently good facility of outflow.

The necessity of a steady state at the beginning of tonography, and a known alteration of that steady state during tonography cannot be overstressed. There have been several misleading reports in the literature by people who intentionally or inadvertently altered the facility or the secretory rate of an eye immediately before or during tonography and drew conclusions regarding the apparent change in facility.

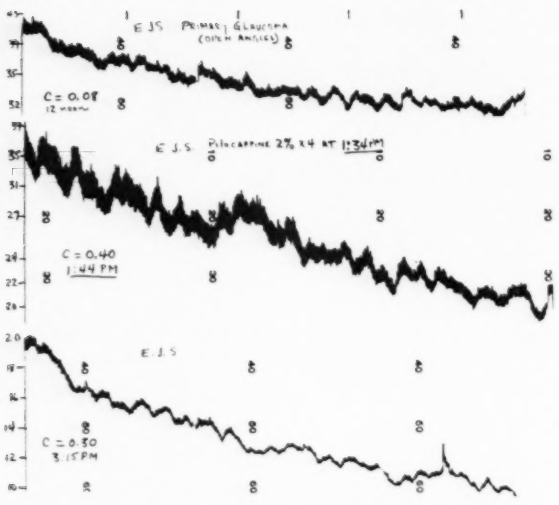

FIG. 1—Tonography during nonsteady state of the eye can produce anomalous results. (Upper) Tonogram characteristic of open angle glaucoma. (Center) Nonsteady state produced by alteration of facility by means of pilocarpine yields elevated pressure in the presence of apparently excellent facility of outflow. (Lower) Approaching steady state under influence of pilocarpine.

Is an eye ever in true equilibrium? Probably rarely. Diurnal changes in pressure are well known, and the existence of such change implies a change in either secretory rate, facility, episcleral venous pressure, choroidal blood volume, or a combination of these factors. However, diurnal alterations in pressure are in terms of hours, tonography is in terms of minutes, and the amount of diurnal change that occurs during the tonogram should be small.

OCULAR RIGIDITY CORRECTIONS

Correction of tonograms for nonaverage ocular rigidity is difficult. The calculations are easy enough, even though in my paper with Dr. Becker I made them seem sufficiently difficult. However, it is the actual measurement of ocular rigidity that is difficult. Dr. Friedenwald emphasized the ease with which large errors may be made in using his two-weight method. Although the combined use of the Goldmann applanation tonometer and Schiøtz (Mueller-Schiøtz) tonometer is potentially more accurate, it is still possible to make large errors. In the first place, both instruments must be very accurate. In a moment I will discuss self-compensation of errors of the tonometer itself in the calculation of the tonogram. Such self-compensation does not apply to the combined use of the applanation and the Schiøtz tonometers.

Applanation tonometry is performed with the patient sitting, but in Schiøtz tonometry the patient reclines, with a consequent shift of systemic blood pressure and episcleral venous pressure.

Correction of the tonogram for abnormal ocular rigidity may help to elucidate the nature of the glaucomatous

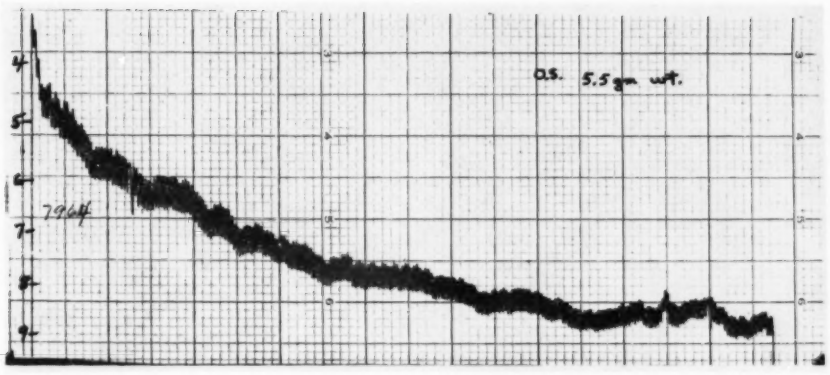

FIG. 2—Rapid drop of pressure in the initial part of the tonogram.

process in the individual eye, to distinguish between decreased facility and hypersecretion, but it does not erase an abnormal set of findings. For example, consider a tonogram, initial scale reading 4.5, final scale reading 6.0 with the 5.5 gm. plunger load. From the table based on average ocular rigidity we obtain the values $P_o = 19$, in the upper ranges of normal, and $C = 0.12$, definitely poor facility. We say that at the moment this patient is compensating for his poor outflow by depressing secretion of aqueous. Suppose that we note that the patient's ocular rigidity is low—0.015, and we recalculate the tonogram allowing for this rigidity. We now find that facility appears better, 0.20, but the P_o is 22 mm. Hg, a more dangerous pressure. We would interpret these data as suggestive that the patient has a facility in low normal range but is secreting aqueous more freely, driving the intraocular pressure up.

The panel thinks it is important to estimate ocular rigidity and to understand the nature of the correction, but that it is not necessary to calculate the correction in most cases.

Several authors (Van Beunigen, Prijot, Moses, Goldmann, Kessler) have published methods for eliminating ocular rigidity as a factor in tonography. Very little has been published on the clinical utility of these methods.

RAPID DROP OF PRESSURE IN THE FIRST MINUTE

Sometimes there is a fairly rapid drop in pressure in the first minute of a tonogram (fig. 2). This phenomenon has been discussed at considerable length. It has been suggested that this fall in pressure represents a slow stretching of the ocular coats, loss of blood volume, or is not all a true fall in pressure but partly represents a viscous flow of the corneal substance and thinning of the cornea under the plunger.

Linnér and Leydhecker have published methods of calculation which eliminate the first minute of the tonogram. However, elimination of the initial reading eliminates the equilibrium pressure, P_o, and so, unless there is an independent estimate of P_o, C cannot be calculated. Calculations by these methods should be labeled something other than "facility,"

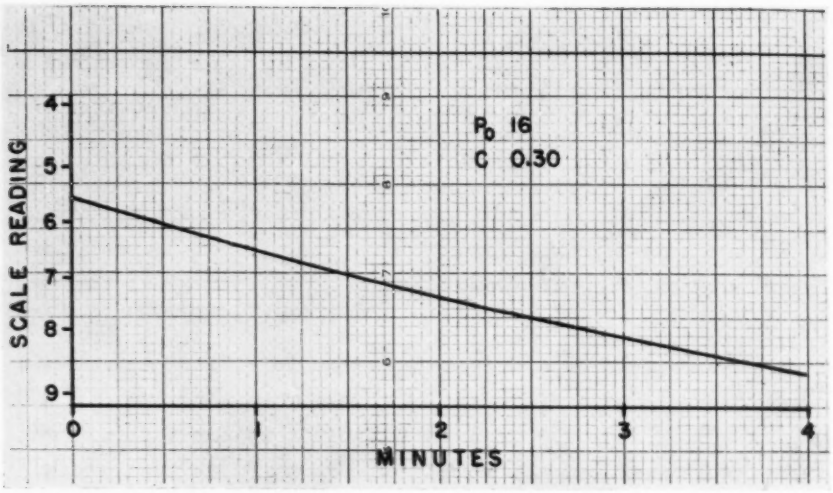

FIG. 3—Ideal tonographic curve calculated from Grant's formula and Linnér's correction for ΔP_v.

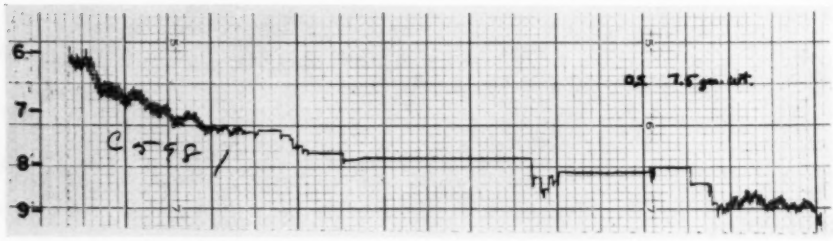

FIG. 4—Uninterpretable tonogram due to sticking of plunger.

as, for instance, Leydhecker's coefficient, or Linnér's index.

According to theory, the tonogram should have a gentle upward concavity. The exact shape will depend on the spacing of the scale units on the recorder paper. Figure 3 represents the ideal curve calculated for $P_o = 16.0$, $C = 0.30$ (5.5 gm. wt.) as plotted on the scale of one of our instruments.

We have found that new technicians tend to transmit their nervousness to the patient and obtain records with the steep initial drop. The best procedure is to discard such tonograms and to repeat the tonography at a later time.

OPERATOR ERRORS

I think the most serious errors made by an operator are (a) using a dirty tonometer, (b) pressing on the eyeball, and (c) tilting the instrument.

The tonometer must be as friction-free as possible, and if the barrel and plunger are not smooth and clean, the tonometer will tend to stick, yielding uninterpretable tonograms (fig. 4).

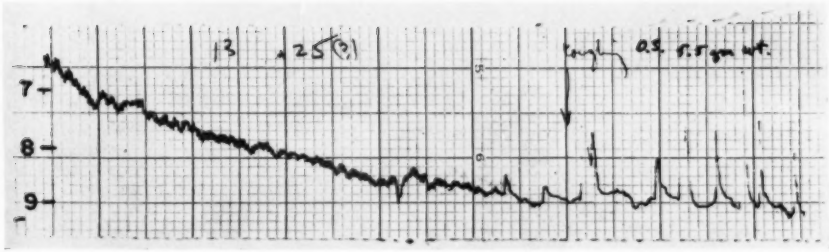

FIG. 5—Effect of cough on tonogram.

It is obvious that the technician must not exert pressure on the globe. The eyelids must be held in such a manner that both patient and operator are comfortable. As in tonometry, the fingers holding the lids must rest on orbital bone and not on the eye. We abandoned lid specula in tonography years ago. The members of the panel feel that the use of specula introduces more artifacts than necessary and we advise against their use in tonography.

Tilting of the tonometer is to be avoided not so much because of the theoretical decrease in effective plunger weight with tilt, which turns out to be quite small for a few degrees of tilt, but because of the rapid increase in friction between plunger and barrel, with tendency of the plunger to stick.

PATIENT ERRORS

The patient's behavior may affect the tonogram in a number of ways. Any change from normal respiration, such as coughing (fig. 5) or breath-holding, affects intrathoracic and hence ocular vascular pressures. If the patient attempts forcibly to close his eyelids, the intraocular pressure can be markedly raised.

A serious problem sometimes arises, particularly in one-eyed patients, who cannot hold their eye steady during tonography (fig. 6).

While these, and other, behavioral difficulties may place the tonogram in question or make interpretation impossible, the nature of the trouble is usually easily noted, and the examiner must be alerted not to place undo weight on such tonograms.

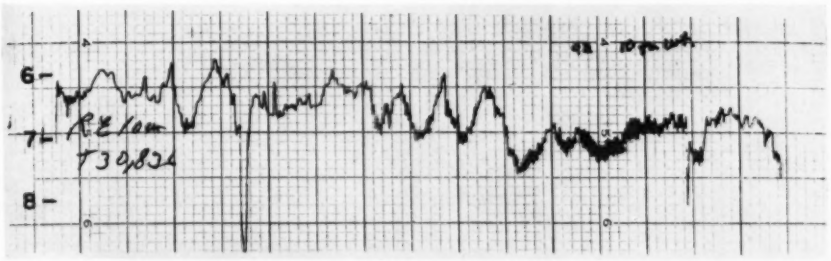

FIG. 6—Poor fixation of the eye during tonography.

INITIAL READING		CHANGE IN SCALE READING		
Po	R (5.5 gm wt)	2.50	3.00	3.50
17	5.00	0.22	0.27	0.33
17	5.25	0.22	0.27	0.33
16	5.50	0.21	0.26	0.32
15	5.75	0.21	0.26	0.32
15	6.00	0.20	0.25	0.31

FIG. 7—Small portion of Becker's table of C (5.5 gm. plunger load). An error of one scale unit in the zero point of the tonometer makes little difference in C.

ERRORS IN THE TONOMETER

Let us suppose that the test block or the footplate curvature of the tonometer is incorrect and that when the tonometer reads "zero" on the test block it should properly read "1." Then scale "5" should be "6," "10" is really "11," and so on. I have purposely taken a very large "zero" error. Now consider a tonogram taken with the 5.5 gm. plunger load, which according to our false scale starts at scale 5 and ends at scale 8 (fig. 7). We read P_o 17, C 0.27; a normal finding. But the tonogram should really go from scale 6 to scale 9. The true values are P_o 15, C 0.25; still a normal finding. As a matter of fact, the facility of outflow is almost unchanged, and we find on inspection of Becker's table that over a fair range, a shift in the scale of the instrument makes relatively little difference in C. Figure 8, taken from Dr. Kronfeld's section of the Decennial Report of the Committee on Standardization of Tonometers of this Academy, shows that a change in the weight of the plunger produces a uniform (linear) shift in the scale reading, similar in this respect to a "zero" error. The variations in weight shown in the diagram are large —far beyond the tolerance limits established by the Committee. Tolerable variations in plunger weight may be expected to give very small changes in P_o and C. The same may be said for the weight of the footplate assembly. This is also true for plunger diameter and edge curve (figs. 9 and 10), since these fac-

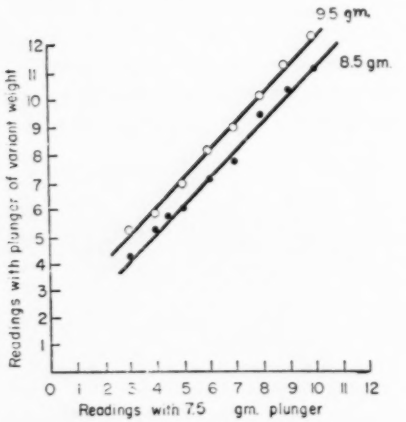

FIG. 8—The effect of variations in the plunger assembly weight upon the tonometric reading. (Kronfeld, Decennial Report, p. 36).

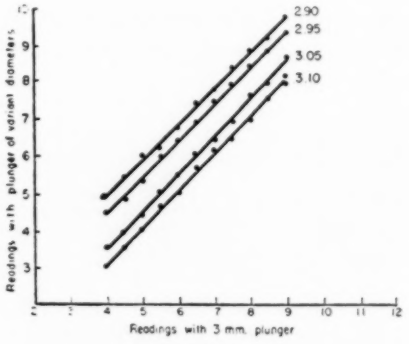

FIG. 9—The effect of variations in plunger diameter upon the tonometric reading. (Kronfeld, Decennial Report, p. 37).

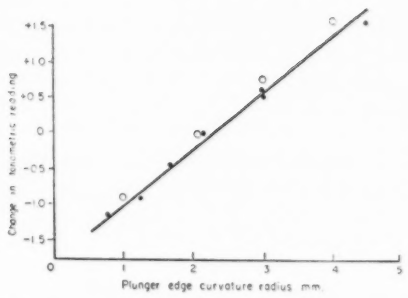

FIG. 10—The effect of variations in plunger edge curvature upon the tonometric reading. The solid dots refer to observed values, the circles to calculated values. (Kronfeld, Decennial Report, p. 37).

tors also effectively shift the zero point of the instrument. I think that it is important to point out that the range of errors of the instrument at all consistent with certification by the Committee standards is ineffective in materially altering the value of C obtained by tonography.

While we recognize many of the errors in the evaluation of the coefficient of facility of outflow by tonography and constantly strive for a reduction in the magnitude of these errors, we are convinced that the inaccuracies as they exist do not invalidate the method.

EXPERIMENTAL TONOGRAPHY

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EXPERIMENTAL tonography has many facets, including many experimental variations in technique and many investigational applications.[6] Experimental tonography is too broad a subject to cover in its entirety in this symposium; we have therefore selected one particular aspect for discussion. Principally, we are to consider the relationship between tonography and quantitative aqueous perfusion of the eye. The aqueous perfusion method offers certain distinct advantages over other experimental methods which might be compared with tonography. Other methods of measurement based on fluorimetry, measurement of outflow pressure, determination of ascorbic acid, or occlusion of episcleral outflow channels with a vacuum cup are known to give estimates of rate and facility of aqueous outflow in human and animal eyes in essential agreement with estimates obtained by tonography,[3,4,6,7,9] but each of these methods is itself indirect and has its own uncertainties. The most direct and most accurate means of measurement is offered by the quantitative aqueous perfusion method. However, in comparing and correlating the results of this method with those of tonography, certain significant differences are to be kept in mind.

In clinical tonography we deal with a living eye having an active vascular bed and tone in the smooth muscle of ciliary body and iris. Yet we require that over all a steady state exist. We proceed on the assumption that the tonographic process itself does not significantly disturb the eye except to increase the rate of outflow.* We are dependent upon estimates of pressure and volume which are inferred from tonometer calibrations, themselves subject to uncertainty and revision, especially in the case of animal eyes.[6,12]

By contrast, in aqueous perfusion of enucleated eyes we can purposely establish a steady state of equal inflow and outflow, and our measurements can be made independent of ocular rigidity.[16] Furthermore, in the enucleated or postmortem eye we obviously have no active innervation and no blood circulation, so we have no variable vascular bed, no flow of blood along with the flow of aqueous in the outflow channels, and therefore no recipient venous back pressure. Moreover, in the ciliary body and iris, there is presumably no activity or tone in the smooth muscle such as there is *in vivo* under innervational or pharmacologic influence. Considering these differences, it is remarkable that measurements of facility of outflow by perfusion postmortem have been found to be as closely in agreement as they have with estimates obtained by tonography *in vivo*.[4,14,16,17]

In aqueous perfusion of the living animal or patient, conditions are much more complex, more closely resembling the conditions encountered in tonography. However, here too, important points of difference are to be considered. First, a needle or cannula must be introduced into the living eye, and it is difficult to

*Recent experimental evaluations utilizing radioactive blood cells have revealed no change of intraocular blood volume during tonography.[5] Possible influence of tonography on aqueous formation is still unsettled.

evaluate how disturbing this may be. In addition, the lens-iris diaphragm is sensitive to changes in volume of the anterior chamber, or to differences in pressure between anterior chamber and posterior chamber, with the result that perfusion into the anterior chamber may deepen this chamber and increase the facility of outflow. To avoid this artifact, it is advisable either to perfuse back of the iris, or to have preliminarily performed an iridectomy for the purpose of maintaining equal pressures in anterior and posterior chambers.

Now, while we keep in mind the differences in conditions, let us see how interpretations of quantitative aqueous perfusion appear to correlate with those of tonography. Long before tonography was developed into a method for quantitative estimation of the facility of outflow in patients, the perfusion method was employed in study of enucleated normal and glaucomatous human eyes.[12] Toward the end of the last century, measurements were made of the rate at which salt solution could escape from the anterior chamber of enucleated eyes at predetermined intraocular pressures, and it was established that glaucomatous eyes offered a greater resistance to outflow than did normal eyes. Subsequently, in tonographic studies on patients, the same conclusions were reached.[11] However, tonographic studies have in addition shown that in comparing normal and glaucomatous eyes it is important to consider for each eye not only its facility of outflow, but also its rate of aqueous formation. In the great majority of cases of glaucoma, tonography indicates that the increase in resistance to outflow is sufficient to account for the observed elevations of intraocular pressure.[11,12] This relationship, which is of particular significance in tonographic diagnosis of glaucoma, will be considered in more detail by Dr. Becker in another part of this symposium.

Continuing with our correlation of information furnished by aqueous perfusion and by tonography, let us next consider certain experimental variables. When the two methods are applied to investigating factors which may influence the facility of outflow in a mechanical fashion, they furnish information which seems to correlate well. For example, pull can be applied experimentally to the scleral spur through the ciliary muscle in perfused eyes by deepening the anterior chamber. This is known from perfusion measurements to cause an increase in facility of outflow.[11] By comparison, miotic drugs, which cause contraction of the ciliary muscle in patients, characteristically, according to tonographic measurements, likewise increase the facility of outflow.[11,12] The mechanical basis of control of outflow appears to be the same in both cases, and experimental evidence localizing the controlling influence to the trabecular meshwork has been obtained by utilizing perfusion in conjunction with either trabeculotomy or detachment of the ciliary muscle, both of which are found to abolish this type of control.[1,12,15]

As another example of correlation of methods, experimental closure of the angle by the iris is readily demonstrated in perfusion experiments to cause a great and reversible increase in resistance to outflow, and clinically a similar reversible increase in resistance is typically found in angle-closure glaucoma by tonography.[11,12]

On the other hand, a fundamental difference between narrowing of the angle by the iris alone and narrowing due to forward movement of the ciliary body has been demonstrated by comparison of aqueous perfusion measurements in enucleated eyes and tonography in patients. When the iris alone bulges forward, as in acute angle-closure glaucoma, the facility of outflow remains normal until

the angle is actually closed.[11] By contrast, when the ciliary body is pushed forward by posterior indentation of the sclera, or by accumulation of fluid between sclera and uvea, the facility of outflow can be gradually decreased without an actual closing of the angle.[8,15] This latter phenomenon is observable after complete experimental removal of the iris, and it is presumably due to compression of the trabecular meshwork, in a sense opposite to the effect of pulling on the scleral spur by deepening the anterior chamber or by contracting the ciliary muscle by miotics.[15] Both phenomena have considerable clinical significance and are responsible for two different types of glaucoma in patients. Recognition of the differences in mechanism provides a clear indication that iridectomy is appropriate for treatment of closure of the angle by the iris alone, but in cases in which glaucoma is caused by forward displacement of the ciliary body, as may follow inward buckling of the sclera and subchoroidal accumulation of fluid, iridectomy alone is ineffectual, and removal of subchoroidal fluid is indicated.

In still more complex clinical situations it has been possible to obtain some comprehension of what is going on only by exploiting all possible methods of investigation. For instance, in study of the mode of action of successful cyclodialysis, we can obtain only a fragment of the necessary information by any one method. Tonographic measurements in the presence of hypotony are ambiguous and difficult to interpret.[6] Aqueous perfusion following enucleation shows no increase in facility of outflow when cyclodialysis is performed.[15] Studies with fluorescein indicate an alteration in permeability and rate of turnover.[10] In this case aqueous perfusion *in vivo* should add a particularly useful piece to the picture. The picture is yet to be completed.

So far, most of our information on hydrodynamic mechanisms in clinical conditions has come from comparison of measurements by different methods during life and postmortem, and often from comparison of similar but not identical eyes. Undoubtedly, aqueous perfusion of human eyes *in vivo* offers the most direct approach for study of many of the clinical problems of ocular hydrodynamics and should afford the most valuable comparison with tonography, especially when both methods are applied to the same eye. We shall hope to see wider application of this procedure with improved techniques in the future. Among the questions which might be settled by quantitative perfusion of patients' eyes is whether increase in intraocular pressure such as induced by tonography does suppress formation of aqueous humor in the human being. No *consistent* answer to this question has been obtained in research on animals; conflicting reports come from different investigators.[2,18]

In certain experimental respects the aqueous perfusion technique has been extended further than have the techniques of tonography. For example, aqueous perfusion has served well in conjunction with experiments in microdissection of the angle of the anterior chamber to establish that some 75 per cent of the total resistance to outflow in enucleated normal human eyes is in the trabecular meshwork, and to show that the pathologic increase in resistance in open-angle glaucoma is due to abnormality in this same tissue, at least in those eyes so far studied.[13,14] Tonographic studies after similar microdissections in patients have yet to be made.

In conclusion, in analysis of ocular hydrodynamics, in investigation of pathogenesis of glaucoma, and in study of physiologic, pharmacologic and surgical influences thereon, for clinical purposes tonography is appropriate. For experimental applications where insertion of a

needle into the eye is permissible, quantitative aqueous perfusion offers advantages in directness and accuracy. Correlation of these two methods has already proved most informative, and is to be highly recommended.

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TONOGRAPHY IN THE DIAGNOSIS OF SIMPLE (OPEN ANGLE) GLAUCOMA

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TONOGRAPHY provides confirmation of the mechanisms of pressure elevation in open angle glaucoma. In addition, tonography offers information about the individual eye which is of considerable help in the diagnosis and evaluation of therapy of its glaucoma status.

The clinical usefulness of tonography stems largely from the many eyes which present flat tonographic tracings but tonometric measurements within normal limits. Empirically, a large number of apparently normal eyes, but for which the tonographic tracings are flat, prove

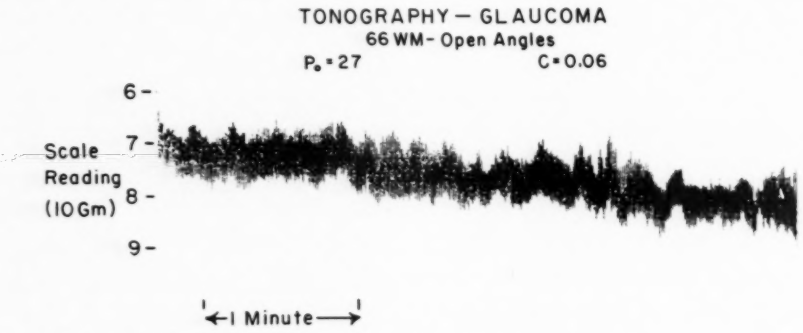

FIG. 1—Tonographic tracing of an eye of a 66-year-old white male with open angle glaucoma.

In the classic instance of glaucoma with open angles, the outflow facility is depressed and the pressure is elevated (fig. 1). Tonography permits us to appreciate the obstructed outflow as the mechanism of the pressure elevation and to confirm the diagnosis. However, in such eyes repeated tonometry is sufficient for recognizing the true glaucoma status. Tonography on these eyes is not essential for making the clinical diagnosis or recognizing inadequate control.

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to be glaucomatous (or inadequately controlled). It is in these eyes that tonography provides valuable clues as to diagnosis or status of control.

There are at least two mechanisms of clinical importance which result in flat tonographic tracings without apparent pressure elevation. The first of these is a decreased ocular rigidity. This occurs especially in myopic eyes, in thyrotropic exophthalmos, following the consumption of a liter of water, after the administration of strong miotics, and after a number of surgical procedures. In such eyes, glaucoma may be detected or its

true status of control appreciated by applanation tonometry, by tonography, or preferably by a combination of the two.

The second mechanism resulting in a flat tonographic tracing in the absence of pressure elevations is a decreased rate of secretion of aqueous humor. Hyposecretion may normalize intraocular pressure in spite of impaired outflow facility. This mechanism may delay damage to the optic nerve and affords a physiologic basis for the clinical use of secretory suppressants. Unfortunately, the spontaneous diurnal (or even prolonged) depressions of secretion are not permanent, and eyes with impaired outflow mechanisms often sustain pressure elevations at times other than those at which they are examined. Thus progression of field loss may occur between visits in spite of apparently normal tonometric measurements made during office visits. The true status in such eyes may be detected by tonography or by frequent pressure measurements at various times during the day and night. From the practical point of view of the clinician's office or tonometric surveys, without routine tonography the diagnosis in these patients is often not suspected until damage to the optic nerve has occurred.

Hypersecretion glaucoma constitutes an extremely rare subgroup of open angle glaucoma. In these unusual cases, tonography confirms the elevated intraocular pressure but reveals that it occurs in spite of reasonably normal outflow facility values. The glaucoma in these instances can be readily detected by tonometry, but its mechanism and suggestions as to appropriate therapy are only appreciated by tonographic measurements.

Considerable data have been accumulated which provide normal values for pressure (P_o) and outflow facility (C). The data for these two measurements have an approximately normal distribution curve. Thus, in a series of over 900 normal eyes, the mean and standard deviation for P_o and C have been calculated (fig. 2). The ratio P_o/C is not distributed in normal fashion but can be converted to a normal distribution by plotting the prevalence of log P_o/C (fig. 3). From such data one can presume that less than 2.5 per cent of the normal population present values more than two standard deviations from the mean (pressure > 21, facility < 0.18, or $P_o/C > 100$), and less than 0.15 per cent vary more than three standard deviations (pressure > 24, facility < 0.13, or $P_o/C > 138$). If such criteria are applied to a group of patients with established glaucoma, open angles, and field loss, our abilities to detect established glaucoma are revealed. From figure 4, it is apparent that with values of more than two standard deviations away from the mean one can detect a little more than half of proved unoperated glaucomatous eyes by pressure measurements alone (over 21 mm. Hg), and some two thirds of eyes by tonography alone (facility less than 0.18). Using the ratio $P_o/C > 100$ permits the recognition of 70 per cent of the same proved cases. A pressure rise of over 7 mm. Hg after the consumption of a liter of water occurs in less than a third of eyes with proved glaucoma. However, the rise from forty to forty-five minutes after intake of water results in considerable elevation of the P_o/C ratio. On the other hand, no significant change in this ratio is noted in normal eyes. Therefore, using the criteria of two standard deviations from the mean for a normal population after consumption of a liter of water, the P_o/C ratio permits the detection of 95 per cent of glaucomatous eyes.

One may choose arbitrarily more rigid criteria for glaucoma diagnosis of three standard deviations from the normal

TONOGRAPHIC VALUES 909 NORMAL EYES

	Mean (M)	Standard Deviation (σ)
P_o (mm Hg)	16.1	2.8
C (μl/min/mm Hg)	0.28	0.05
log P_o/C	1.75	0.13

FIG. 2—Mean values and standard deviations for pressure (P_o), outflow facility (C) and log P_o/C for a series of 909 normal eyes.

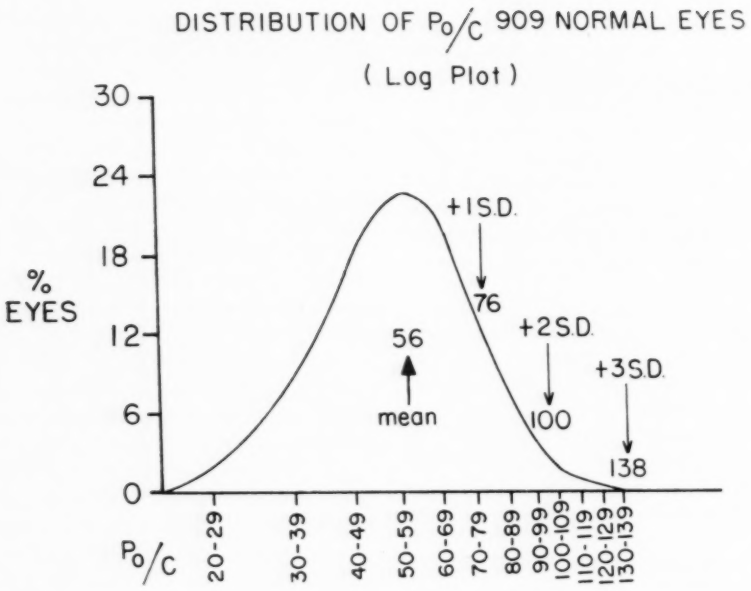

FIG. 3—Normal distribution for log P_o/C.

PREVALENCE OF TONOGRAPHIC FINDINGS IN EYES OF NORMAL, GLAUCOMA PATIENTS, AND GLAUCOMA FAMILIES

Finding ($M + 2\sigma$)	Normal (909 Eyes)	Glaucoma (250 Eyes)	Family (220 Eyes)
$P_0 > 21$	2.5%	54%	15%
$C < 0.18$	2.5%	65%	20%
$P_0/C > 100$	2.5%	73%	21%
After H_2O			
8 + mm rise P_0	2.5%	30%	3%
$P_0/C > 100$	2.5%	95%	31%

FIG. 4—In each group, the number of eyes with values in excess of two standard deviations from the mean for a normal population.

PREVALENCE OF TONOGRAPHIC FINDINGS IN EYES OF NORMAL, GLAUCOMA PATIENTS, AND GLAUCOMA FAMILIES

Finding ($M + 3\sigma$)	Normal (909 Eyes)	Glaucoma (250 Eyes)	Family (220 Eyes)
$P_0 > 24$	0.15%	35%	8%
$C < 0.13$	0.15%	43%	11%
$P_0/C > 138$	0.15%	50%	14%
After H_2O			
$P_0/C > 138$	0.15%	74%	20%

FIG. 5—In each group, the number of eyes with values in excess of three standard deviations from the mean for a normal population.

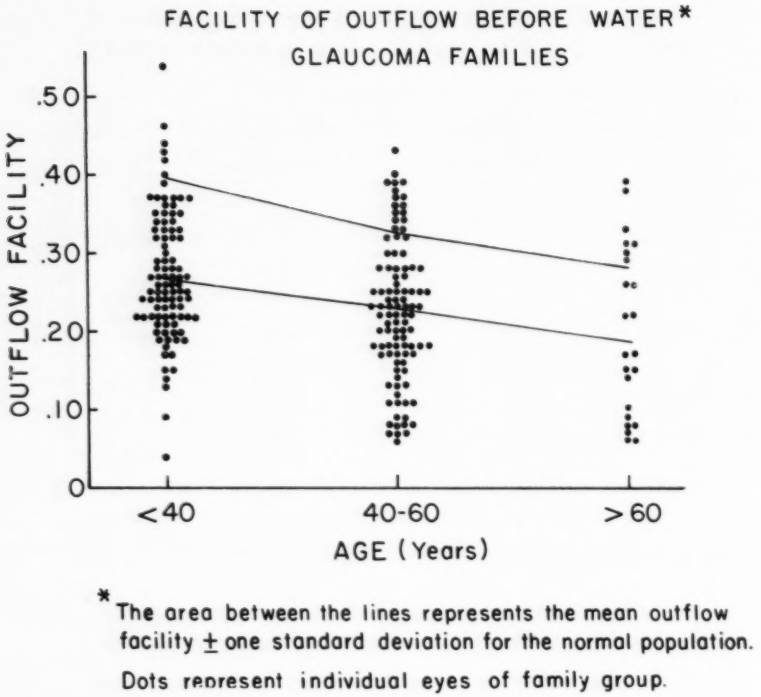

FIG. 6—Individual values for outflow facility *before* water drinking in the families of patients with glaucoma.

mean, so as to include less than 3 in 2000 normals (fig. 5). Under these circumstances, pressure measurements (over 24 mm. Hg) detect only a third of proved glaucoma with open angles, and outflow facility estimates (less than 0.13) only some 40 per cent of eyes. However, after water drinking a P_o/C of greater than 138 occurred in almost three fourths of these same glaucomatous eyes.

It is well established that open angle glaucoma may be inherited and that families of glaucomatous patients have a high prevalence of the disease. Applying the above tests to the immediate families (siblings, parents and children) of glaucoma patients should indicate a considerably higher prevalence of abnormalities than in the normal population. Furthermore, in such a family population one can hope to detect the earliest stages of the disease process before the onset of established glaucoma with cupping and field loss. In a series of 220 eyes with open angles studied in the close relatives of glaucoma patients, it was found that a decreased outflow facility was very prevalent both before and after water drinking (figs. 6 and 7). Further-

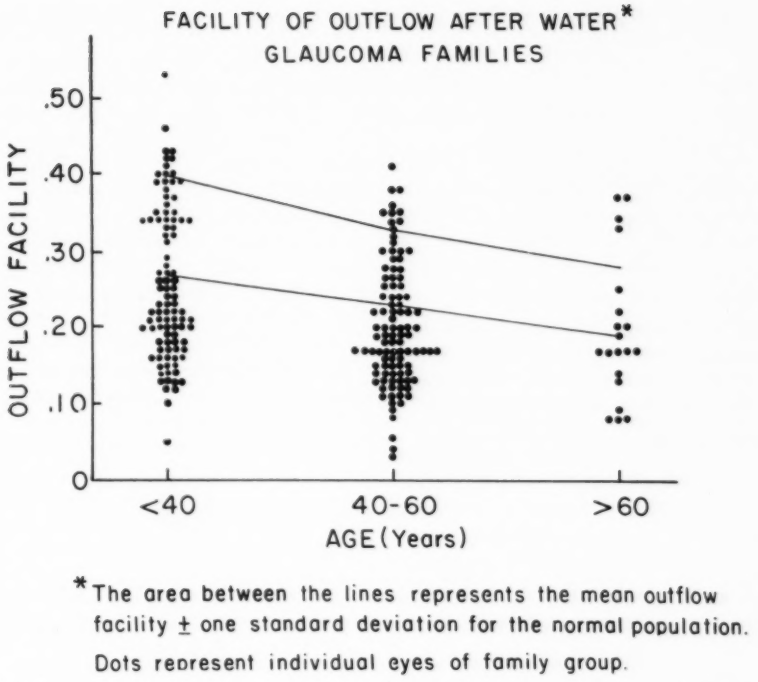

FIG. 7.—Individual values for outflow facility *after* water drinking in the families of patients with glaucoma.

more, at values that were found in 2.5 per cent of the normal population (2 S. D.) "abnormal" pressures (over 21 mm. Hg) occurred in one in seven eyes of the family group; an outflow facility less than 0.18 was recorded in one in five eyes of this series; and a P_o/C ratio of greater than 100 after water drinking was calculated for almost one third of these same eyes (fig. 4). Even at a most significant level of three standard deviations, the P_o/C was still abnormal (over 138) in one fifth of eyes of glaucoma relatives (fig. 5).

The study of individual patients in families with a strong family history of glaucoma not only permits the early detection of abnormalities, but also offers an opportunity to study the progression of changes over a period of years. In our experience the first abnormality noted is an elevation of the P_o/C ratio after water drinking. Subsequently there is spontaneous elevation of the P_o/C ratio, and about this time outflow facilities less than 0.18 are found. At a later time intraocular pressures over 21 mm. Hg occur. Still later, outflow facility values less than 0.13 are observed and are followed by pressure elevations to over 24 mm. Hg. Many years may intervene before field loss and cupping are recognized.

FOUR YEAR FOLLOW UP ON 40 "NORMAL" EYES WITH INITIAL $P_o/C > 100$

Finding	Incidence
$P_o > 21$	22 (55%)
$P_o > 24$	13 (33%)
$P_o \geq 30$	6 (15%)
$C < 0.18$	29 (73%)
$C < 0.13$	19 (48%)
8 + mm rise P_o after H_2O	4 (10%)
Field loss	5 (13%)

FIG. 8—The occurrence of elevated pressures and reduced outflow facilities over a period of four years in eyes which initially had no defects, pressures less than 21 mm. Hg, but P_o/C values > 100 after water drinking.

A question naturally arises regarding the significance of a P_o/C ratio over 100 after water drinking in a population of patients with otherwise normal eyes. A four-year follow-up was made on 26 patients, eight patients with proved open angle (simple) glaucoma in their contralateral eyes, twelve patients with a family history of glaucoma and open angles but no evidence of the disease themselves, and six patients with no history or family history of glaucoma (fig. 8). These patients provided 40 eyes which appeared entirely normal to all initial tests and studies except for the finding of a P_o/C greater than 100 following the consumption of a liter of water. All of these eyes had open angles, initial pressures less than 21 mm. Hg, and no evidence of cupping or field loss. It is apparent that more than half of such eyes at some occasion in the four-year period had pressure measurements that were over 21 mm. Hg. Furthermore, one third of the eyes had isolated pressure measurements of over 24 mm. Hg, and 6 of the 40 eyes had pressure measurements as high as 30 mm. Hg. Almost three fourths of these eyes had outflow facilities less than 0.18 on some occasions, and almost half had recorded facilities less than 0.13. In the four-year follow-up, field loss (Bjerrum scotomas) occurred in five eyes (13 per cent) of this small series.

It is thus apparent that although tonography may not be essential for the recognition of classic open angle glaucoma, it provides a useful short cut and valuable adjunct to other methods for the diagnosis and evaluation of therapy of the disease. At the present time an elevation of the P_o/C ratio appears to offer a simple means for the early detection of glaucoma as well as the recognition of its inadequate control.

TONOGRAPHY IN THE MANAGEMENT OF GLAUCOMA

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In discussing tonography in the management of glaucoma, it should be emphasized that this is no attempt to discredit the classic guides which have been used through all the modern history of treatment of glaucoma. Control of the intraocular pressure, careful attention to visual field loss, and repeated observation of changes in the discs are invaluable in the management of glaucoma, and are, as they have always been, reliable guides to the course of glaucoma. However, it is our feeling that tonography offers a new tool of unusual predictive value, which makes possible more sensitive management of simple glaucoma than has previously been possible, and more logical and accurate choice of operation in closed angle glaucoma than is possible without the use of this technique.

SIMPLE OR OPEN ANGLE GLAUCOMA

It goes without saying that our major therapeutic problem in glaucoma concerns the management of simple or open angle glaucoma. The aim of treatment in this ubiquitous disease is, as it has always been, the retention of visual fields. Our limited understanding of the mechanisms involved in progressive loss of visual fields has made it essential for us to concentrate our therapeutic efforts on the reduction of the intraocular pressure. In general, adequate control of intraocular pressure has led to success in our efforts to preserve visual fields. However, it appears that the new goal of normalization of the facility of aqueous outflow, or C value, offers rewards not previously attainable by attention to control of the intraocular pressure alone.

It has now been demonstrated by several investigators that medical control of simple glaucoma may be better achieved by utilization of tonographic criteria for control than by attention to ocular pressure alone. Data from a long-term study of early glaucoma done at Bowman Gray School of Medicine have demonstrated that, first, visual fields are almost always retained when the C value is normalized and, second and conversely, visual field is usually lost in the presence of usually abnormal C values. The correlations are much less exact if normalization of pressure is the sole criterion for control. Using for normalization of the C value, a value maintained during therapy at .18 or more, and for normalization of pressure, an intraocular pressure of 21 mm. of mercury or less, the data may be presented in the following study involving 185 eyes. These are all cases of early glaucoma which have had adequate follow-up for at least one and one-half years and up to nine years. Visual fields could be studied satisfactorily on all these eyes. Visual fields were intact or showed minimal loss at the time of detection of all cases, and loss incurred on therapy during this study was in most cases very early field loss, detected on the 1/1000 or 2/1000 white isopter.

It may be observed that when the facility of outflow is maintained at .18 or above, visual field is retained in 89 per cent, or almost nine tenths of the cases,

From the Bowman Gray School of Medicine.

but when it is usually below .18, field is retained in only 18 per cent, or less than one fifth of the cases (fig. 1). Similar correlation of visual field retention with pressure level is not so impressive, with about half the cases retaining field in this group even with elevated pressure (above two standard deviations from the normal mean), and with almost one third of the cases maintaining apparently normal pressure but nevertheless losing visual field.

THERAPEUTIC CRITERIA AND VISUAL FIELDS
EARLY GLAUCOMA - MIOTIC THERAPY

	Retained Visual Field	Lost Visual Field
C-Value usually > 17	98 (89%)	13 (11%)
C-Value usually < 18	13 (18%)	61 (82%)
Pressure usually > 21	49 (52%)	46 (48%)
Pressure usually < 22	62 (69%)	28 (31%)

FIG. 1—Data on 185 eyes with early glaucoma followed one and one half to nine years. (Department of Ophthalmology, Bowman Gray School of Medicine)

Becker has presented an extensive study of the prognostic value of tonography in the control of simple glaucoma, involving 250 eyes treated by miotics, from the Glaucoma Clinic of the Washington University School of Medicine. These data have pointed out that continued control, as defined by lack of visual field loss or by pressure which never exceeds 24, is directly related to the initial C value. When the C value was better than .17, 84 per cent of cases showed continued control for three years; whereas, in the same series an intraocular pressure below 22 mm. of mercury resulted in only 66 per cent of cases showing such control.

If the ratio of the initial pressure to the facility of outflow (P_o/C) was below 100, perhaps the most successful control possible was found, with control being maintained in better than 90 per cent of the cases. The results of these data are summarized in figure 2, prepared from data by Becker. The favorable prognostic significance of a C value within two standard deviations of the normal mean or of a P_o/C less than or equal to 100 is clearly evident from these data.

PROGNOSTIC SIGNIFICANCE OF TONOGRAPHIC VALUES ON MIOTIC THERAPY

Initial Tonographic Criteria		Percentage Successfully Controlled *
Po < 22	} 2 standard deviations	66 %
C value > .17		84 %
Po/C ≦ 100		91 %

* Successful control defined as no pressure rises to over 24 mm. Hg. and no loss of visual field on miotic therapy for over three years. (from Becker)

FIG. 2—Data on 250 eyes with three-year follow-up, from Becker. (Department of Ophthalmology, Washington University School of Medicine)

Ballintine has followed a group of cases in which the intraocular pressure was rigidly controlled without regard for C values. He has found striking correlation between the C value and the degree of retention of visual fields. For this study, 280 eyes having proved glaucoma (that is, pressure above 30 mm. Hg Schiøtz on several occasions, or pressure over 30 mm. Hg on one occasion plus a P_o/C greater than 100 after water drinking) were used. These were all regulated at a pressure level below 25 mm. Hg initially, and if the pressure went above 25, they were discarded. Therapy was adjusted to maintain pressure above 18, but at or below 20 mm. Hg Schiøtz, as nearly as possible, without regard for C value. If therapy was not followed as prescribed, if the pressure went above 25, or if follow-up visits were inadequate, the case was discarded. Therapy was by miotics alone. Visual field loss, which was defined as a reproducible Bjerrum scotoma or a peninsular baring of the blind spot to 1.7/1000

Correlation of Visual Field Loss to C-Value and Pressure

72 eyes followed from 1 to 6 years in pressure range 16-25mm.Hg. (Ballintine)

Average C	Number Observed	Number Field Loss	% Field Loss
0-.12	30	13	43
.13-.17	20	9	45
.18 and above	22	1	4
Total	72	23	
Pressure Range			
>20	38	9	23
<21	34	14	41

FIG. 3—Data from Ballintine. (Western Reserve University School of Medicine)

white isopter or larger, was then correlated to the C values observed and to pressure levels maintained.

From this study 72 eyes were obtained in which the pressure never exceeded 25 mm. Hg Schiøtz during periods of follow-up of at least one and up to six years. As indicated in figure 3, thirteen out of thirty eyes with an average C value between 0 and .12 lost field, nine out of twenty eyes with an average C value from .13 through .17 lost field, whereas only one out of twenty-two eyes with an average C value of .18 or above (within two standard deviations from the normal mean) lost field. From these same 72 eyes, correlation of field loss with average pressures above 20 mm. Hg Schiøtz and average pressures below 21 mm. Hg Schiøtz (fig. 3) is essentially meaningless, with actually more field loss observed in the lower pressure levels within this range, despite the very rigid regulation of pressure (fig. 4). The essential identity of pressure data in eyes losing field with those not losing field is quite evident here, unlike the distinct differences in C value for eyes losing and for eyes retaining visual fields. From this it is evident that visual field retention correlates much more closely with the level of C value than with pressure level.

PRESSURE DATA

72 eyes followed 1-6 years

Average I.O.P. 20.7mm. Hg.

	Field Loss	No Field Loss
Maximum	25	25
Minimum	17	16
Median	20	22
Average	20.2	21

FIG. 4—Data from Ballintine. (Western Reserve University School of Medicine)

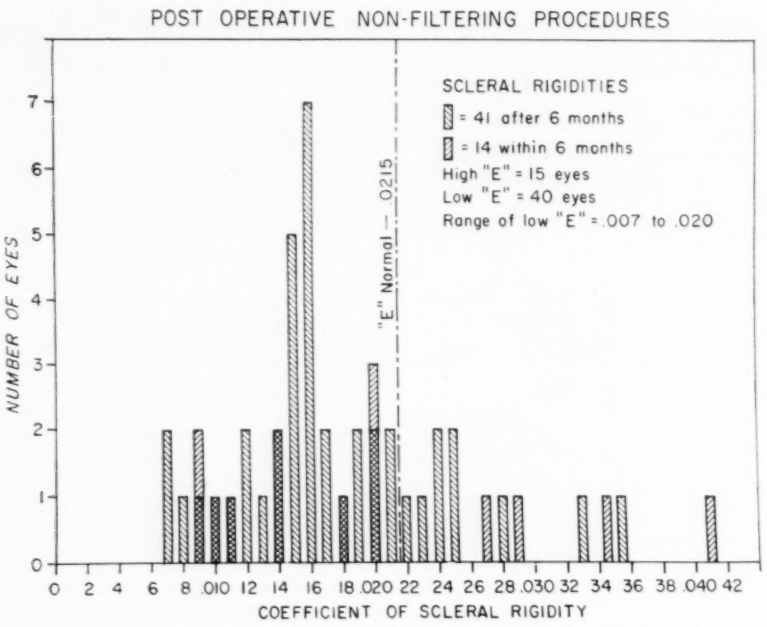
POST OPERATIVE NON-FILTERING PROCEDURES
SCLERAL RIGIDITIES
= 41 after 6 months
= 14 within 6 months
High "E" = 15 eyes
Low "E" = 40 eyes
Range of low "E" = .007 to .020
"E" Normal — .0215
NUMBER OF EYES
0 1 2 3 4 5 6 7
0 2 4 6 8 .010 12 14 16 18 .020 22 24 26 28 .030 32 34 36 38 .040 42
COEFFICIENT OF SCLERAL RIGIDITY

FIG. 5—Data on surgical cases from Department of Ophthalmology, Bowman Gray School of Medicine.

Effect of Drug Therapy

In considering the value of tonography as an aid to the management of glaucoma, an understanding of the effects of drug therapy on the facility of outflow must be obtained. Perhaps the most important actions of the miotic drugs in so far as glaucoma is concerned are, first, to raise the C value or increase the facility of outflow and, second, to lower the intraocular pressure. Indeed, if a poor facility of outflow in an equivocal case suspected to be simple glaucoma is improved by the use of 1 per cent pilocarpine, support is given to the diagnosis of glaucoma.

Among the outstanding recent improvements in drug therapy of simple glaucoma has been the renewed appreciation of the value of epinephrine. Tonographic studies point out that the effect of epinephrine in lowering the intraocular pressure is primarily due to a reduction in aqueous flow which averages approximately 37 per cent. However, an unexpected additional result, which has recently been pointed out chiefly by Ballintine, has been an improvement in the facility of outflow, which is usually delayed but which may occur early.

The use of aqueous inhibitors, such as Diamox and other carbonic anhydrase inhibitors, also has been demonstrated to lower pressure primarily by reducing aqueous flow. The use of Diamox alone appears to produce an average reduction in flow of 50 per cent, but if it is combined with levo-epinephrine, the flow can be reduced by better than 65 per cent in most cases. To date, no long-term im-

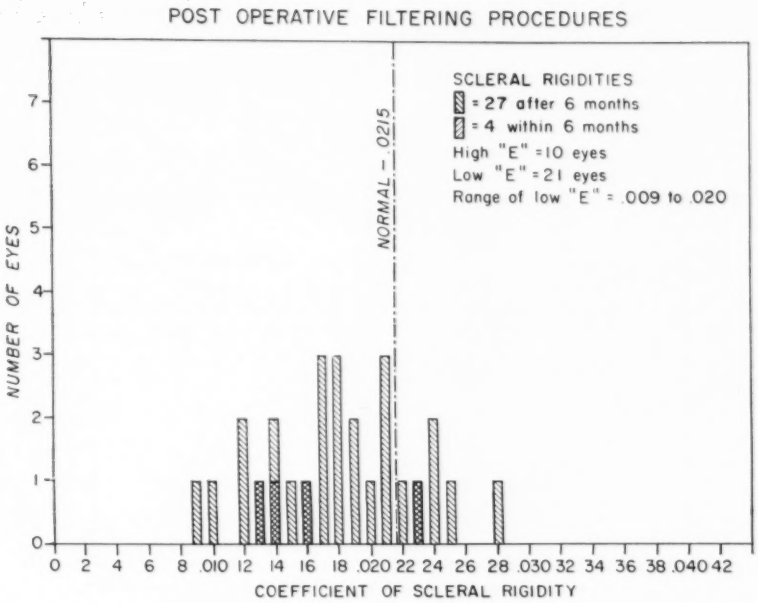
POST OPERATIVE FILTERING PROCEDURES
SCLERAL RIGIDITIES
= 27 after 6 months
= 4 within 6 months
High "E" = 10 eyes
Low "E" = 21 eyes
Range of low "E" = .009 to .020
NORMAL — .0215
NUMBER OF EYES
0 1 2 3 4 5 6 7
0 2 4 6 8 .010 12 14 16 18 .020 22 24 26 28 .030 32 34 36 38 .040 42
COEFFICIENT OF SCLERAL RIGIDITY

FIG. 6—Data on surgical cases from Department of Ophthalmology, Bowman Gray School of Medicine.

provement in facility of outflow on carbonic anhydrase inhibitors alone has been documented.

In the infrequent form of open angle glaucoma which we refer to as hypersecretion glaucoma—and which can only be placed in such a classification by tonographic evaluation of the increased rate of aqueous flow — it would appear that the most logical therapy would be use of aqueous inhibitors, either alone or in conjunction with miotics.

Tonography and Surgery in Simple or Open Angle Glaucoma

It should be particularly emphasized that the facility of outflow alone should never be used as an indicator for surgical treatment in simple glaucoma. The need for surgical therapy should remain, as it has always been, dependent upon inability to prevent loss of visual fields by the most effective medical therapy which can be found. Often visual fields can be retained for a very considerable period after the C value and pressure have not been controlled by miotics alone, if inhibitors of aqueous flow are used in addition. Therefore, it should be emphasized again that surgical treatment is indicated only if visual fields cannot be retained.

In the surgical treatment of simple glaucoma, tonography may prove useful as a follow-up examination after a filtering operation. If the intraocular pressure is normalized and *low* following filtering or some other surgical procedure for simple glaucoma, the C value is usually high. On the other hand, if the intraocular pressure is low and the C value is also low, it should be suspected

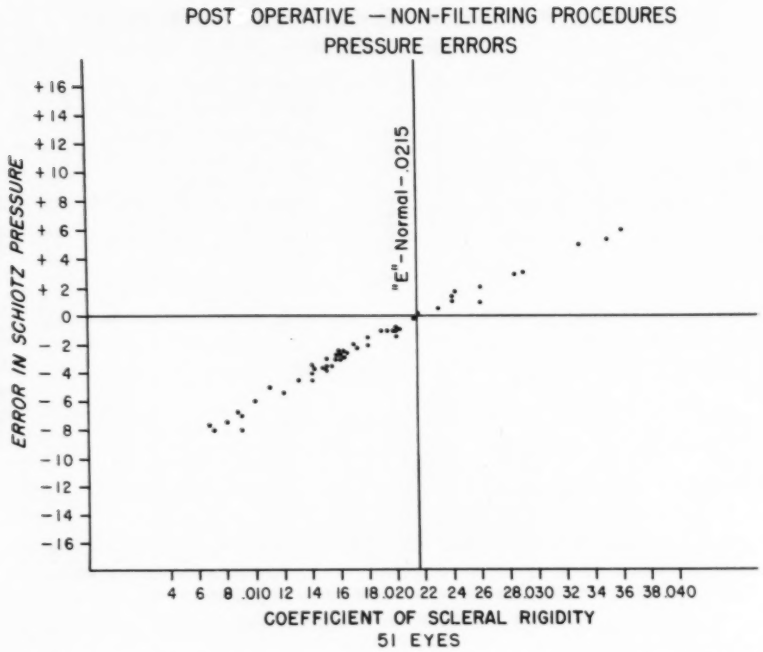

FIG. 7—Errors in Schiøtz pressure measurements resulting from abnormal postoperative scleral rigidities.

that low ocular rigidity is present, or that hyposecretion is present along with poor outflow facility. Either of these may lead to an appearance of good control while the pressure actually is still elevated or outflow still poor. Lowered rigidity, with *false* normalization of pressure, may account for some instances of progressive visual field loss in the presence of apparently good surgical control. That such low scleral rigidity values are frequent after surgical procedures, filtering or otherwise, is suggested by data I have compiled at Bowman Gray School of Medicine. These data, based on repeated paired applanation and Schiøtz readings, show that any intraocular surgery, filtering or otherwise, may produce altered, and most often low, scleral rigidity. These alterations may produce startlingly false values for the intraocular pressure, which may give a false sense of security unless the low rigidity is recognized. From bar graphs representing scleral rigidity measured on glaucomatous eyes on which nonfiltering surgical procedures such as cataract extractions and iridectomies had been performed, the marked preponderance of low rigidities, both within six months from the time of the operation and after six months may be observed (fig. 5). In figure 6, similar preponderance of low rigidities after filtering surgery is indicated. In figures 7 and 8, the degree of error in the apparent Schiøtz pressures resulting from these abnormal rigidities is indicated by scatter graphs on the pressure errors in these same postopera-

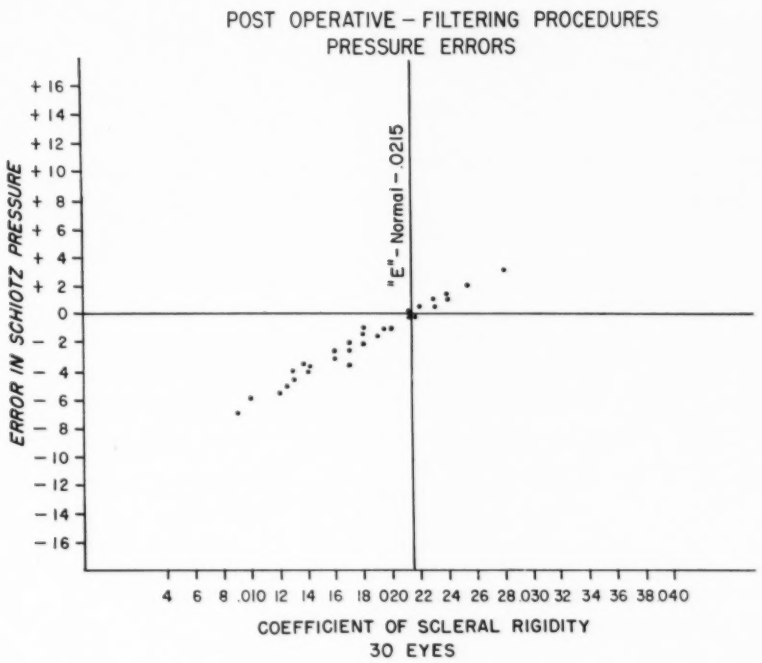

FIG. 8—Errors in Schiötz pressure measurements resulting from abnormal postoperative scleral rigidities.

tive cases. It may be seen that there are far more falsely low values than accurate or falsely high values, and that the falsely low pressures were as much as 8 mm. Hg lower than the true pressures as measured by applanation. Even in the absence of availability of applanation, however, such abnormal rigidities may be suspected where discrepancies between pressure levels and C values cast doubt on the true status of control in such cases.

CLOSED ANGLE GLAUCOMA

In the handling of closed angle glaucoma, tonography may be an invaluable aid in determining the functional state of the meshwork after angle closure episodes and, consequently, in determining the appropriate surgical treatment which should be used in any given case. With acute congestive angle closure episodes, while gonioscopically the angle is completely, or virtually completely, closed, it is inevitable that the facility of outflow will be low while the pressure is elevated. However, after the pressure has been normalized by the use of miotics, or miotics combined with aqueous inhibitors in some cases, the angle still may be gonioscopically so narrow or so nearly closed that certainty about the presence of organic change in the angle structures, whether they be anterior synechias or more subtle damage in the trabecular meshwork itself, may be difficult or impossible to determine. In such instances, and particularly in cases in which it has been necessary to use Diamox or other aqueous inhibitors to bring the pressure

down, the use of tonography may be of critical importance in determining what type of surgical procedure may be expected to give lasting control of the pressure.

If the angle is not apparently open after the pressure has been normalized by medical methods, but if a good facility of outflow is found on tonography, it can be expected that a simple iridectomy will be curative, since it will prevent future angle closure episodes and since the trabecular meshwork apparently is capable of normal function. On the other hand, if the angle appears to be partly open or even virtually completely open after medical therapy has normalized the pressure following the acute congestive episode, and the facility of outflow is still poor, then it may be expected that the outflow mechanism is damaged or that simple glaucoma is concurrently present with the angle closure glaucoma. Consequently, a simple iridectomy may not produce the desired cure, and a filtering procedure may be indicated. If the C value is borderline, or if other factors in the clinical picture suggest that medical therapy may succeed after angle closure is made impossible, it may be entirely justifiable to do a peripheral iridectomy in the hope that if the outflow and/or pressure remains somewhat abnormal postoperatively, the residual glaucoma can then be controlled by miotics. Strong miotics or epinephrine may be safely used after such an iridectomy. Tonography also adds value to mydriatic provocative tests in subacute or interval angle closure glaucoma if, when the pressure rises following the administration of mydriatics, the angle can also be demonstrated to be partly or completely closed and the facility of outflow to be subnormal after previously being normal (fig. 9).

TONOGRAPHIC CRITERIA

Closed Angle Glaucoma

After Medical Therapy of Acute Episodes of Angle Closure

T $\leq$ 21 C $\geq$ .18	Peripheral prophylactic iridectomy indicated regardless of angle appearance
T $\leq$ 21 C > .10 C < .18	Iridectomy may be done, with possible need for subsequent medical R_x, including strong miotics or epinephrine
T $\leq$ 21 C < .11	Filtering procedure usually advisable, regardless of angle appearance

With mydriatic provocative: C value falling below .18 or significantly from previous levels, with angle at least partly closed = Angle Closure Glaucoma

FIG. 9—Summary of tonographic criteria used in selection of operation in closed angle glaucoma and in confirming the diagnosis of closed angle glaucoma.

SUMMARY

It would certainly seem reasonable that the management of glaucoma can be achieved on a more logical basis if the status of the facility of aqueous outflow is known both before and during therapy. Tonography gives us this information for the first time, and by evaluation of tonographic data we should be able to assess the adequacy of medical therapy in simple glaucoma better and earlier than has been possible heretofore. In closed angle glaucoma it should make possible more accurate selection of the appropriate operation for each individual case.

ON APPLANATION TONOMETRY

THEODOR F. A. SCHMIDT, M.D.
BERN, SWITZERLAND
BY INVITATION

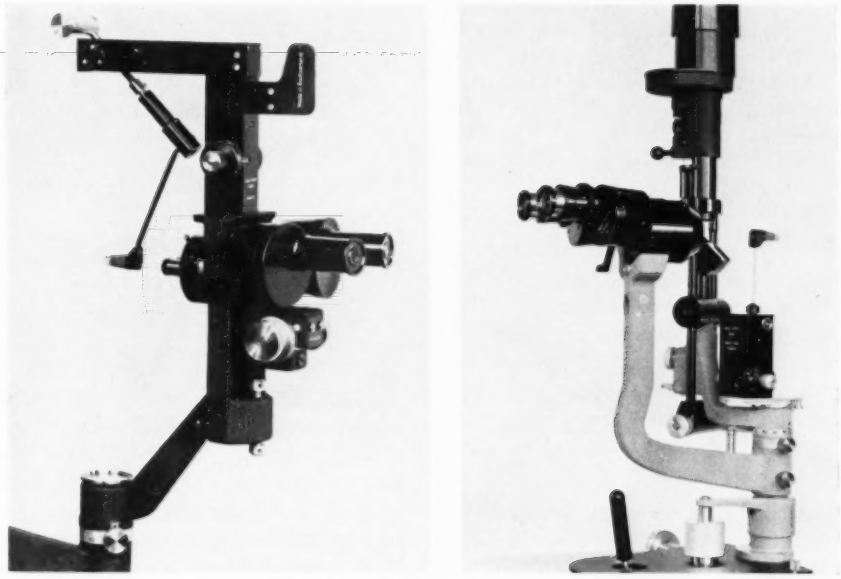

FIG. 1—(Left) The Goldmann applanation tonometer on the older model of the Haag-Streit slit lamp. (Right) The applanation tonometer on the new Haag-Streit slit lamp 900.

FOR some years now the Goldmann Applanation Tonometer, which is used in conjunction with a slit lamp, has been available to the ophthalmologist. Two models of the applanation tonometer are available, one used with the old model of the Haag-Streit slit lamp (fig. 1, left), the other one with the new slit lamp 900 (fig. 1, right). Either one or the other model (with a suitable adapter) can be used on practically all slit lamps on the market. Many authors have considered the theory and practice and the advantages and disadvantages of the applanation tonometer. I shall summarize here only briefly what is known so far.

The applanation tonometer is based on the following principle: measurement of the force necessary to flatten a small corneal surface of constant size. The main advantage of the applanation tonometer is the small volume displacement (of about 0.5 $mm.^3$) during the measurement. This has the result that the intraocular pressure of the untouched eye is raised by only 2.5 per cent, and

From the University Eye Clinic, Bern, Switzerland; Prof. Hans Goldmann, Director.

Presented at the Sixty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1960, Chicago.

the most important result of this is that the rigidity of the eye and the radius of the cornea affect the result so slightly that the measured pressure can be regarded as equal to the original intraocular pressure of the untouched eye.

The question arises, How important is it to know the scleral rigidity? In other words, how often does the actual intraocular pressure vary significantly from the value measured with one plunger weight of the Schiøtz tonometer and the value given in the table for P_o based on the assumption of average scleral rigidity (E=0.0215)?

It may be that there are no great variations in the ocular rigidity coefficient when measuring normal individuals. From the standpoint of an eye surgeon, who likes to detect and treat glaucoma, the knowledge of variations in scleral rigidity coefficients of normal individuals is not very useful.

We have measured the ocular rigidity of 384 glaucomatous eyes using the applanation tonometer and the Schiøtz tonometer with the 7.5 gm. plunger load. These included operated and unoperated eyes, both old and young glaucoma patients, as seen at our clinic. Scale reading 5.3 with the 7.5 gm. Schiøtz weight corresponds to a P_o of 25, according to Friedenwald's 1955 table, but—as table I shows—only 121 eyes out of 384 glaucomatous eyes, i.e., 31.5 per cent, would actually have an intraocular pressure between 23.5 and 26.5 mm. Hg. The table shows also that in a large percentage of glaucoma patients serious errors would be made if reliance were placed on the measurement with a single plunger weight and ocular rigidity were not considered. In our experience, *in cases of myopia and particularly in cases after operation, ocular rigidity is lowered and the Schiøtz tonometer indicates a pressure lower than actually exists.*

TABLE I

VARIATIONS IN P_o VALUES DUE TO VARIATIONS IN RIGIDITY COEFFICIENTS

VARIATIONS FROM THE TABLE VALUE 25 mm. Hg = 5.3/7.5 g	%
< ± 1.5 mm. Hg	31.5
> ± 1.5 mm. Hg	68.5
> ± 3.0 mm. Hg	43.0
> ± 4.5 mm. Hg	26.0
> ± 6.0 mm. Hg	14.0
> + 7.5 mm. Hg	8.0
> + 9.0 mm. Hg	4.0
> +12.5 mm. Hg	1.0

Large variations of the corneal curvature are less frequent than variations of ocular rigidity so that, in general, considerable errors of the P_o value using one Schiøtz weight are less influenced by corneal curvature than by ocular rigidity, which can be demonstrated from a statistical study of Leibowicz[9] on corneal curvature of 10,330 eyes. At a scale reading of 5.3/7.5 gm. in 7.9 per cent of the population the intraocular pressure would be more than 26 mm. Hg and less than 24 mm. Hg instead of 25 mm. Hg; in other words, the error would be greater than ± 1 mm. Hg. In 1.1 per cent the error is greater than ± 2 mm. Hg. These numbers are valid only for eyes with a mean value of ocular rigidity. If the ocular rigidity varies from this mean value, then greater or smaller total errors are to be expected. However, it is useful to know the corneal curvature in any case of microcornea or megalocornea when the Schiøtz tonometer is used, but I would like to insist that ocular rigidity and corneal curvature have negligible influence when the applanation tonometer is used, so that it is not necessary to measure them.

In the case of all tonometers which

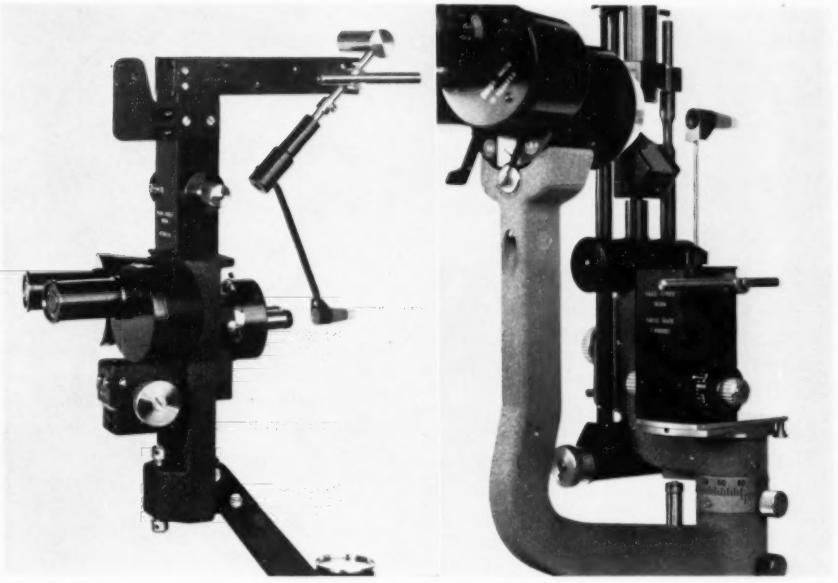

FIG. 2—(Left) The calibration weight and the correction ring on the applanation tonometer. (Right) The calibration weight on the new model of applanation tonometer.

cause more deformation of the globe, in particular in the case of the Schiøtz tonometer, the rigidity of the eye and the radius of the corneal curvature have a decisive effect on the result of the measurement. They may appreciably falsify the results of measurements if they are not taken into account.

Since the intraocular pressure is raised only slightly during the measurement with the applanation tonometer, there is no tonographic effect if the measurements are repeated. If, nevertheless, pressure changes are observed in the course of a series of measurements, this must be due to other causes.

The applanation tonometer has also the advantage of enabling one to read the results directly in millimeters of mercury.

Independently of us, Moses[10-12] found that the results of the measurements agreed excellently with the manometer readings found in the course of calibration tests on freshly enucleated human eyes. Calibration tables, which may change every few years, as in the case of the Schiøtz tonometer, are therefore not required for the applanation tonometer.

Another important advantage of the applanation tonometer is the simplicity of its design. There are only two construction factors which may become sources of errors, apart from the friction: (1) the balance which establishes the pressure required for flattening a certain area of the cornea, and (2) the measuring prism which measures the flattened cornea.

The balance enables the pressure to be varied from zero to 8 grams, corresponding to an intraocular pressure of 0 to 80 mm. Hg. The tonometer for the

older Haag-Streit slit lamp has a spring, which establishes the pressure. This spring has been stretched 20,000 times, and in operation does not reach the limit of elasticity. The force of the spring can always be checked by means of the calibration weight (fig. 2, left). Any small error can be corrected by adjusting a small correction ring.

The applanation tonometer for the new slit lamp 900 utilizes a weight instead of a spring. In this case also a calibration weight is provided for checking (fig. 2, right).

Theoretically, error could arise during the measurement of the flattened area. The area, which must always be of the same size, is given by the diameter of the flattened cornea. This diameter is measured by means of two prisms mounted inside the measurement prism (fig. 3) which causes the picture to be

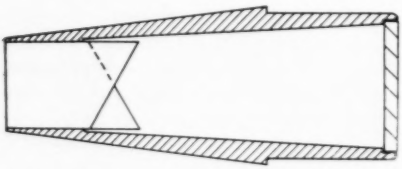

FIG. 3—The measuring prism of the Goldmann applanation tonometer.

halved, and displaced by 3.06 mm. The action of the prisms can be checked with the aid of a gauge (fig. 4) consisting of two lines spaced 3.06 mm. from each other. The displacement is subject to a tolerance of ± 0.05 mm., corresponding to an error of about ± 3 per cent in the measurement. Even this small tolerance is not fully utilized, a fact which I have proved to myself, so that the error is practically zero. There is no displacement of the prisms, because the prism surfaces are ground on a solid piece of material.

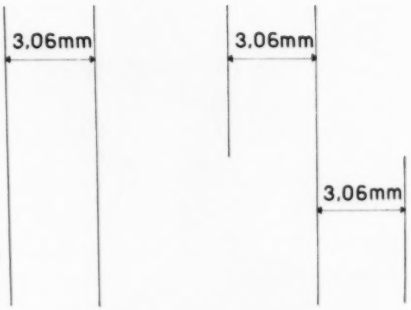

FIG. 4—Gauge for checking the prism. Diagram of appearance of gauge seen through a correct prism.

The reason for using the prism for measuring the diameter of the small flattened area is that only one point is under observation, namely, the point at which the two semicircles of the fluorescein ring overlap (fig. 5). It will be clear that accurate measurements of a very small area are made possible because only one measuring point has to be observed and this by a 10 times magnification of the slit lamp microscope.

The fact that the instrument error of the applanation tonometer is practically zero means that the examining physician can be quite certain of the accuracy of the results of the measurements. Even a standardized Schiøtz tonometer may have an instrument error varying between plus 3 and minus 3 scale divisions, as calculated by Albrecht and associates.[2] Although this extreme case rarely happens in practice, deviations of plus 1 or minus 1 scale division are by no means rare even in accurately calibrated instruments. Thus, Dr. Armaly of Iowa City obtained averages of 16.5, 17.8 and 18.9 mm. Hg when he carried out comparative measurements with three electronic tonometers.

Finally, we have to consider also the reading error, which affects the result of the measurements.

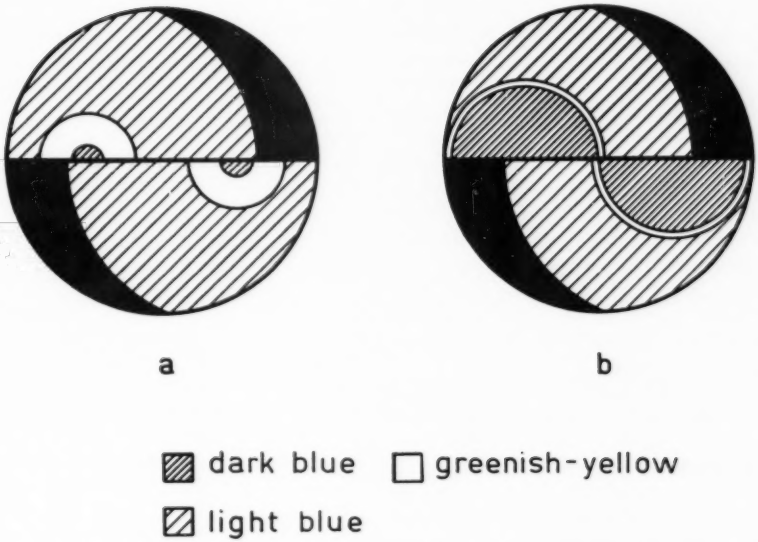

FIG. 5—(a) Diagram of the microscopic appearance of the prism in contact with the cornea when a small amount of force is applied. (b) Sufficient force is applied to the cornea to establish an applanation diameter of 3.06 mm. at which diameter 1.0 gm. of force corresponds to 10 mm. Hg.

As regards the Schiøtz tonometer, Rohrschneider and Kuchle[13] calculated a measuring error of ± 1 scale division for the individual reading. If several measurements are taken, and the average is found by calculation, the reading error is reduced, but if numerous measurements are carried out with the Schiøtz tonometer, there is also a tonographic effect, so that the number of measurements to be carried out consecutively should not exceed 3. If the applanation tonometer is used, quite a large number of measurements can be carried out without any tonographic effect, so that the error of the reading can be reduced, by calculating the average of numerous individual readings, to less than ± 0.5 mm. Hg.

The one condition is, however, that one must use the applanation tonometer correctly. Various sources of errors have to be eliminated during the measurements. This matter cannot be discussed in detail here, but I shall be pleased to point out these sources to interested parties by way of practical demonstrations.

In the meantime we have continued our research work in different directions: (1) the influence of various applanation diameters in human eyes and (2) applanation tonometry on animal eyes. I will speak briefly about the last point, because it may be interesting to research workers.

We did manometer experiments on enucleated eyes of dogs, cats and rabbits to find the relationship between the intraocular pressures and tonometer readings. We carried out measurements with different sizes of the flattened area: 3.06, 3.5, 4, 5, 6, 6.5 and 7 mm. of diameter at different manometer levels up to 50 mm. Hg.

The result is that the so-called Imbert-Fick law, which is the basis for applanation tonometry, cannot be applied without corrections to the applanation tonometry of the examined animal eyes. The Imbert-Fick law states that the pressure in a sphere filled with liquid and surrounded by an infinitely thin membrane is measured by the minimum counterpressure required to flatten the membrane to a plane. This law presupposes that the membrane is without thickness and without rigidity and no other forces interfere in the measurement.

The cornea of humans and animals is not without rigidity. Moreover there are other forces than that of the tonometer which have to be considered, such as capillary forces between the surface of cornea and the surface of the measuring prism.

On the human eye the elastic forces are equalized by the capillary forces when an area with a diameter of about 3 mm. is flattened, as was demonstrated by Goldmann.

In the measurement of animal eyes, the mentioned forces do not balance—at least not with flattened areas with a diameter up to 7 mm. We cannot use such large areas, for they are accompanied by large volumetric displacements and the ocular rigidity errors are similar to those experienced with the Schiøtz tonometer. For that reason we tried to find the relationship between the intraocular pressure and the weight required to flatten a small area.

We found quite a linear relationship between intraocular pressure and applanative force. Different equations for each animal and each size of flattened area were developed. Our experiments, which soon will be published in extenso, suggest the use of a 4 mm. prism instead of a 3.06 mm. prism when measuring dog, cat or rabbit eyes. The displaced volume then amounts to about 1.5 mm.^{3} Table II shows the equations. The resulting calibration curves do not differ greatly, and they are practically identical for cats and rabbits (fig. 6).

TABLE II

EQUATIONS CHARACTERIZING THE RELATIONSHIP BETWEEN THE MEASURING WEIGHT AND INTRAOCULAR PRESSURE FOR RABBITS, DOGS AND CATS WHEN A FLATTENED AREA WITH A DIAMETER OF 4 MM. IS USED

Rabbits:	Tension = 6.48 × Weight + 4.02
Dogs:	Tension = 6.33 × Weight + 2.26
Cats:	Tension = 6.30 × Weight + 4.41

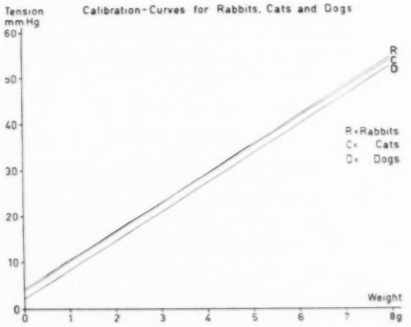

FIG. 6—Calibration curves for rabbits, dogs and cats measured with a flattened surface of 4 mm. of diameter.

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BILATERAL CONGENITAL GLAUCOMA

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THE term *congenital glaucoma* applies to those cases of increased intraocular pressure in which the decreased aqueous outflow is due to a defective embryologic development of the eye.[4] A study of congenital glaucoma from the standpoint of histology is extremely valuable when the eyes are obtained early in the course of the disease before degeneration, atrophy and fibrosis have resulted from the ravages of uncontrolled elevated intraocular pressure.

The embryologic development of the filtration angle is not one of atrophy of the mesoderm but rather of a specific cleavage resulting in separation of the iris and ciliary body from the trabecular meshwork. Allen and his colleagues[1] in their clear, detailed, and beautifully illustrated article showed that in the process of development of the filtration angle there was differentiation and orientation of the cells, cleavage between the iris and ciliary body and trabecular meshwork, associated with unequal fetal growth of the parts, and cavitation resulting in the formation of the normal angle. Their specimens demonstrated clearly that it was not a process of atrophy.

Congenital glaucoma appears either at birth or shortly thereafter. Maumenee[3] reports that in a series of 362 patients glaucoma was present at birth in 47 per cent and appeared during the first six months of life in 31 per cent. By this one sees that approximately three fourths of the patients suffer from the disease sometime during the first six months of their life. This makes early diagnosis imperative.

The early symptoms are photophobia, epiphora and blepharospasm. The symptoms tend to be cyclical in nature, adding to the confusion in the early period of the disease. Later there is edema of the epithelium, haziness of the stroma, stretching of the cornea with enlargement, ruptures in Descemet's membrane, and finally permanent opacification of the cornea. The principal pathologic changes at first are primarily in the anterior segment. In patients under three years of age, the eye is somewhat elastic and will respond to the abnormal increase in intraocular pressure by stretching. It is only late in the disease that the optic nerve becomes cupped and destroyed by atrophy. Maumenee[3] states that photophobia and epiphora in any child under two years of age is strongly indicative of increased intraocular pressure and emphasizes the importance of immediate referral to an ophthalmologist for examination under deep general anesthesia.

Shaffer[5] stresses the importance of proper education of pediatricians and general practitioners, who see many of these children during the early period

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From the Good Samaritan Eye Pathology Laboratory, Good Samaritan Hospital, Portland, Oregon.

I am indebted to William Spaulding, M.D., Tacoma, Washington, who made available these valuable specimens.

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and who, because of lack of knowledge of congenital glaucoma, treat the patients erroneously for ocular inflammation or occlusion of the tear ducts. It is imperative that information be disseminated among these practitioners so that the cases of congenital glaucoma will be found in their early stages and treated before the cornea is stretched and destruction of the canal of Schlemm occurs.

Barkan,[2] whose brilliant contribution brought about the widespread use of goniotomy, felt that he could see the iris inserting into a semitranslucent, cellophane-like, shagreened surface and this in turn to the ring of Schwalbe. Maumenee[3] after extensive studies of clinical and histologic specimens stated that gonioscopy alone was not adequate. In general, the infant tissues are more translucent and the trabecular meshwork has a more cellophane-like appearance. Iris processes are more common. The iris tends to arise closer to the scleral spur than in the adult. Maumenee[3] was unable to see evidence of the cellophane-like membrane reported by Barkan. In a few cases he found adhesions of the anterior leaf of the iris to Schwalbe's line or to the trabecular face. This adherence resulted in a membrane-like covering of the angle. In some cases he noted that the iris appeared to rise from Schwalbe's line or seemed to be inserted into the scleral spur or the most anterior portion of the ciliary body. Maumenee also found that in a majority of these cases, at least two thirds of the trabecular meshwork appeared to be exposed to aqueous. By the use of jugular compression he was able to demonstrate that the canal of Schlemm was present and could be filled with blood, and that the cause of the glaucoma was not due to the absence of the canal of Schlemm. He also pointed out that the longitudinal fibers inserted into the trabecular meshwork and produced an abnormal function so that the trabecular spaces failed to open and that there was also a compression of the canal of Schlemm.

Normally the longitudinal fibers insert into the scleral spur and with contraction tend to aid the opening of the spaces and the canal of Schlemm. This observation seems to be extremely valuable in view of the fact that many of the early glaucoma specimens which have been studied show a limited obstruction of the trabecular face by adherence of the iris to it. Clinically the use of miotics, such as pilocarpine, did not seem to aid facility of outflow, suggesting an abnormal function of the longitudinal fibers of the ciliary body.

CASE REPORT

Colored male child, full term at birth, began rubbing eyes at age 4 months. Mother stated that they became red and watered badly. The corneas then appeared hazy. Tension was taken under anesthesia and found to be 45 in the right eye, 50 (Schiøtz) in the left. Goniotomy was performed on the nasal side of each eye. The tension was apparently controlled without medication for approximately ten days. It then rose to 60 in the left eye and 30 in the right. Similar goniotomies were performed on both eyes, using areas which had not been previously opened. A goniopuncture was included in the operation performed on the left eye. The tension in each eye remained well controlled without medication for a period of one month. At that time the child suddenly died of congenital heart disease, complicated by pneumonia.

Grossly, both eyes appeared to be of normal size and shape and the corneas were not distended. Small scars could be seen where the goniotomy knife had entered in the performance of surgery.

Histopathology

Right Eye (fig. 1), imbedded in paraffin. Although there is considerable variation in the sections throughout vari-

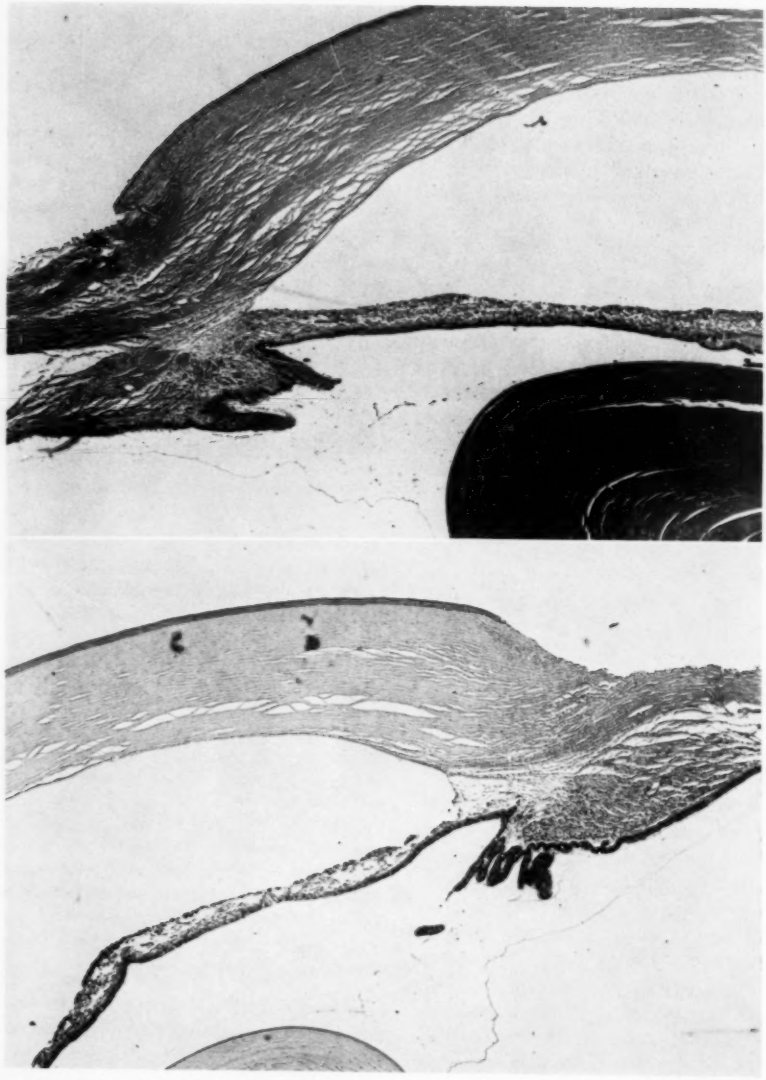

FIG. 1—Right eye, showing incomplete cleavage and forward attachment of meridional fibers on trabecular face.

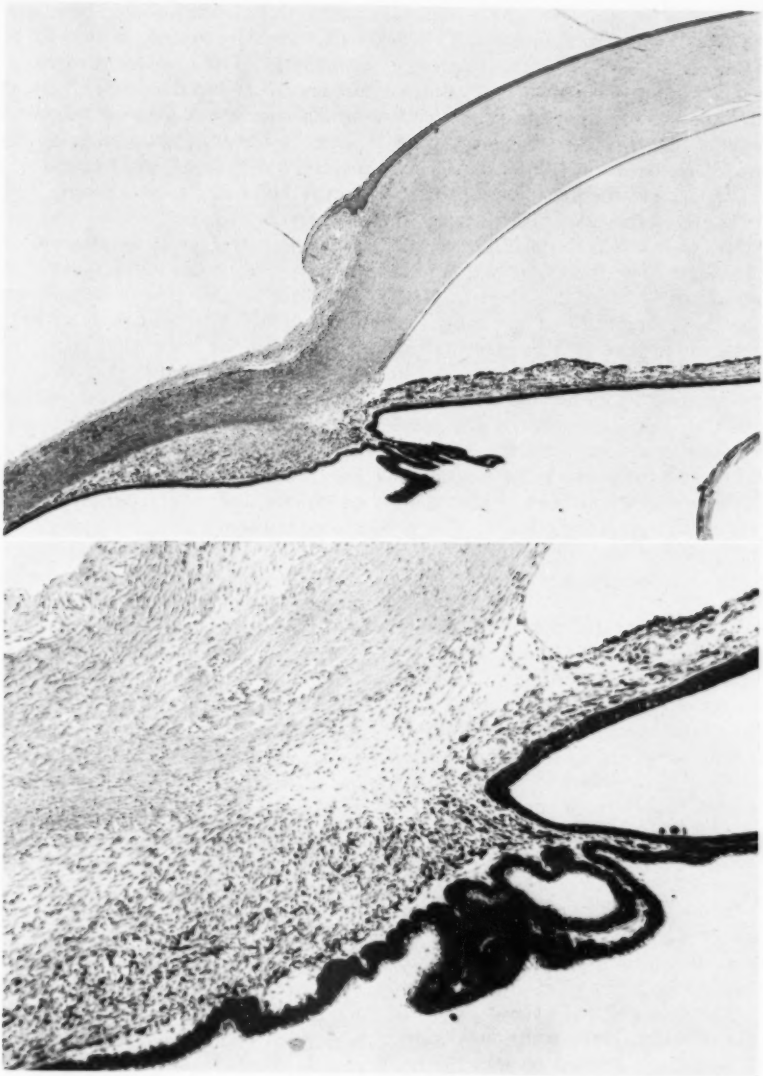

FIG. 2—Left eye, showing incomplete cleavage and forward attachment of meridional fibers on trabecular face.

ous portions of the eye, the principal pathologic changes are found in the filtration angle. In some regions the angle shows almost no tendency to separation of the iris and the ciliary body from the trabecular meshwork and scleral spur. There is also poor development of the scleral spur, and the meridional and circular fibers of the ciliary body appear to be attached to the trabecular fibers forward to the spur. The spur not only is poorly developed but also appears to be compressed laterally. The canal of Schlemm is present in all slides studied, but it shows considerable variation in size depending upon the amount of compression that has existed. In one small area there was an artificial cleavage separating the iris from the trabecular meshwork which had been produced by the tip of the goniotomy knife. The remainder of the eye, including the optic nerve, appears to be normal.

Left Eye (fig. 2), imbedded in celloidin. The left eye shows the same variation and the same type of anomaly in the development of the angle as seen in the right eye. There appears to be even more firmness in the adherence of the iris and ciliary body to the trabecular meshwork and scleral spur. The same forward attachment of the longitudinal muscle fibers to the trabecular meshwork is apparent, with compression of the spur and collapsing of the canal of Schlemm. The scleral spur is poorly developed. In one area a fresh scar exists corresponding to the point of goniopuncture. This was not patent and probably not functioning. There was considerable small round cell infiltration into the iris and ciliary body. The remainder of the globe including the optic nerve appears to be normal.

COMMENT

When one considers the pathogenesis of congenital glaucoma, it becomes apparent that the case should be recognized early and treatment instituted before stretching of the anterior segment and degeneration and fibrosis of the trabecular meshwork and canal of Schlemm have resulted. The goniotomy, when performed early, seems to achieve what nature has failed to do: proper cleavage and freeing of the iris and ciliary body from the trabecular meshwork. This permits the proper development of the scleral spur and proper attachment of the longitudinal fibers to it rather than forward to the trabecular cells. Barkan strongly recommended that the incision be shallow, not to exceed one-fourth millimeter. Maumenee[3] recommends that the knife enter at the level of Schwalbe's line, be inserted only a fraction of a millimeter, and not be permitted to open more than two fifths of the angle. There still exists controversy between able authorities whether or not the goniotomy should be done under the contact lens. In the skillful hands of Otto Barkan, goniotomy under the lens was carried out with a high degree of perfection. It appears to me that when one must enter at the point of Schwalbe's line and not penetrate deeper than one-fourth millimeter, it must be done with extreme precision. The technique set down by Barkan using his lens and knife is still of great value in performing the goniotomy operation.

SUMMARY

A case of bilateral congenital glaucoma was presented to demonstrate the pathologic change existing in the filtration angle. This case was diagnosed and treated in the early stages before stretching and degeneration of the anterior segment had developed. The canal of Schlemm was still present. Following goniotomy the tension remained normal for one month without treatment. This child suddenly died of congenital heart disease complicated by pneumonia.

It is exciting to contemplate studies to be made in the future which will demonstrate more clearly the interrelationship between congenital glaucoma and other angle anomalies and general collagen defects occurring throughout the body, all resulting from a common cause.

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BACTERIAL ALLERGY OF THE EYE

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IN ophthalmology, perhaps more than in any other branch of medicine, allergy, in the past decades, has come to be accepted as a major etiologic mechanism. An increasing number of apparently unrelated ocular affections are now best explained on the basis of hypersensitivity. This is not surprising in view of the fact that the eye and its adnexae offer a unique opportunity to observe and study, both clinically and experimentally, fundamental allergic reactions to an extent not possible elsewhere in the body.

However, since certain forms of ocular allergy such as those due to anaphylaxis, atopy (hay fever), and contact reactions (to drugs, cosmetics, and other chemicals) are readily recognizable, such categories are undeservedly highlighted. As a result, the great importance of bacterial allergy in the causation of ocular disease is underestimated, because the reaction incited is less immediately dramatic and more difficult to diagnose. Similarly, the emphasis now belatedly being given to autogenous allergy as an etiologic mechanism (long appreciated by ophthalmologists in relation to sympathetic ophthalmia and phacoanaphylactic endophthalmitis) merits special attention. Actually, in terms of ocular disability and the serious visual damage that may ensue, hypersensitivities arising on a microbiallergic basis, as well as those occurring due to a possibly related autoallergy, constitute the most important forms of ocular allergy the ophthalmologist encounters.

This paper will survey the various ocular manifestations of bacterial or microbial allergy; that is, hypersensitivity to all types of microbial products (bacterial, fungal, parasitic and, possibly, viral). I have coined the adjective *microbiallergic*[17(a)] to describe reactions of this type. There is considerable evidence that microbiallergic reactions may occur in every part of the eye and the eyelids. Although the mechanism can usually be explained as a direct antigen-antibody response, as in other allergies, it is sometimes less clear-cut or direct, and seems to require an intermediate agent. In such instances, it is possible that the concept of autosensitivity can serve as an explanation, since there is some evidence that auto-allergy can result when foreign proteins from microorganisms, toxins, or other products, unite with a tissue hapten after breakdown of the tissue protein-hapten complex.[14]

THE BASIS FOR THE MICROBIALLERGIC REACTION

It is now an established fact that many of the harmful effects that microorganisms can exert in the human body are manifestations of allergy. Over and above the direct toxic nature of microbial infection, allergy may convert a harmless protein product of bacterial disintegration into a violent and lethal poison. In ophthalmology, such bacterial allergy, especially to staphylococcal products, is of great importance in diseases of the eyelids, conjunctiva, cornea

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and sclera; hypersensitivity to products of the Streptococcus and the tubercle bacillus appear to play an important role in uveal diseases and in scleritis.

A distinction must be made between microbial allergy and immunity. When toxins are absorbed in the course of infection, immunity occurs if the body overcomes the invading organisms. Such immunity is expressed by the formation of antitoxins. Similarly, microbial products evoke the formation of different types of immune antibodies, such as agglutinins and precipitins. In a perhaps oversimplified sense, the immunity thus achieved is a protective process, while allergy is an inflammatory and necrotic process which is essentially harmful.

In all of these cases, allergy develops as a result of infection, and it is manifested by injury or necrosis of cells in contact with the organism. In chronic infections, the pathogenesis of the disease may be profoundly altered by change in the reaction of the host to the parasite.[11] This is illustrated by the caseation of tubercles, and by the development of gummas in tertiary syphilis, after sensitization has become marked. The sensitized person will react specifically to suitable antigens, generally proteins, from the causative organism, by an inflammatory reaction to a skin test. These tests are often of diagnostic value, but the sensitization persists indefinitely and is not necessarily evidence of active infection.

While true anaphylaxis identical to that caused by serum has been produced experimentally in animals from large amounts of dead typhoid and tubercle bacilli, as well as from yeast, bacterial anaphylaxis as such does not appear to occur in human beings. According to the dosage and route administration, hypersensitivity in man manifests itself in local, focal or systemic reactions. Skin reactions from intradermal injections of microbial products generally are of two types: (1) most of the time, a delayed allergic response (tuberculin-type), and (2) relatively rarely, the immediate wheal-type response. The latter is seen more commonly in reactions to molds and Monilia than in reactions to bacteria or trichophytin. I have found, however, that staphylococcal products often cause immediate reactions of this type in particularly hypersensitive individuals. A third form of reaction, eczematous or epidermal-contact type in character, may also occur in microbial dermatitis along with the other two reactions. Passive transfer tests are negative. Histamine does not appear to play an important role in the reaction. Humoral antibodies are not demonstrable in the sera of patients; instead, antibodies seem to be bound to the cells.

While most observations, understandably, have been made with bacterial and fungal products, enough studies on parasitic agents (helminths) have been done to demonstrate that what might be called infestation allergy to either the tissue protein or excretory products of the parasites occurs as well. Such helminthic reactions are sometimes of the immediate variety. The blood eosinophilia that occurs with parasitic infestation is suggestive of an allergic mechanism. Skin tests utilizing helminthic products as antigens are of value in the diagnosis of parasitic infestations. Reactions similar to anaphylaxis have been elicited in dogs and guinea pigs by the initial intravenous injection of deproteinized extract of Ascaris. Rupture of an echinococcus cyst may cause anaphylactic shock.

Relatively little has been proved in the field of viral allergy. Diagnosis of lymphogranuloma venereum is aided by a Frei skin test; skin reactions occur in revaccination for smallpox and are useful in mumps.

Hypersensitivity to bacterial toxins

plays an important part in allergic conditions. Burky's staphylococcal experiments,[3] confirmed by others, indicate that such toxins may be sensitizing factors even in the absence of allergy to the bacterial protein itself. This work is largely the basis for our considering microbiallergic conjunctivitis as an entity. Furthermore, it should not be inferred that only those bacteria that elaborate exotoxins may incite microbiallergic reactions. Experiments with Escherichia coli endotoxin and other toxic products of gram-negative bacteria[2] indicate that their effects are the result of a specific immunologic reactivity similar to tuberculin sensitivity, and that they can elicit reactions in patients through both toxic and immune mechanisms. Endotoxin has been identified as a lipopolysaccharide present in, but easily separated from, the washed bacterial cells of gram-negative bacteria, but not found in gram-positive bacteria.

MICROBIALLERGIC CONJUNCTIVITIS

Bacteria, fungi and helminths appear to be capable of inciting microbiallergic conjunctivitis. The status in regard to viruses is not clear.

Bacteria

There is very little evidence that bacteria other than the Staphylococcus play an important role in the production of allergic conjunctivitis. Apparently a combination of two factors operates in the pathogenesis of this condition: (1) chronicity, with the opportunity for repeated reinfection, and (2) the formation of antigenic exotoxin or other sensitizing substances. Most bacteria that produce potent, but not necessarily allergic, exotoxins, such as the diphtheria and tetanus bacilli, cause acute processes. Only the staphylococci appear to fill both requirements in this respect. It must be emphasized that microbiallergic conjunctivitis may occur without the presence of, or previous exposure to, what we consider pathogenic organisms. Even nonpathogenic bacteria can bring about an allergic state in certain individuals. This fact would explain the occurrence of microbiallergic conjunctivitis where only the so-called non-toxin-producing Staphylococcus albus is found.

Recent studies of bacteriophage typing of Staphylococcus aureus by Locatcher-Khorazo and Gutierrez[7] may offer, by inference, additional confirmation of the role of bacterial allergy in staphylococcal infections. Although many phage types were isolated by these authors from normal eyes, it was rare to find more than one strain in the eyes of any single individual, and the same phage type was present in the nose as well. Staphylococcal ocular infections, including unilateral postoperative infections, were caused by the same type that was present in the normal eye of the same individual. In eight postoperative infections the same phage type found before operation was responsible. Repeated ocular cultures show that the same phage type of staphylococci persisted for months, even after antibiotic therapy.

Although others have reported microbiallergic conjunctivitis to streptococcal and gonococcal products, I have not encountered such instances.

In a detailed study of 60 instances of all forms of conjunctival allergy, other than those due to contact allergy, investigated at the Fondation Ad. de Rothschild in Paris, Morault[8] found that seven cases were due to bacterial allergy. According to Morault, the responsible organisms, which in some instances were multiple, were pneumococcus in 5 cases; Streptococcus hemolyticus in 5; Staphylococcus aureus in 3; Streptococcus viridans, colibacillus, and Pseudomonas pyocyaneus each in 1.

TABLE I

DIFFERENTIATION OF CHRONIC TYPES OF ALLERGIC CONJUNCTIVITIS[17(b)]

	CHRONIC ATOPIC CONJUNCTIVITIS	MICROBIALLERGIC CONJUNCTIVITIS DUE TO STAPHYLOCOCCUS
History	Familial allergy frequent Other associated allergies	Familial allergy infrequent No associated allergies
Allergens	Pollens, danders, inhalants, foods	Bacterial products, mainly Staphylococcus toxin
Allergic response	Immediate	Delayed
Symptoms	Burning, irritation, and often severe itching	Burning, irritation, and often marked dryness
Discharge	May be mucopurulent	Watery, no pus
Lid margins	Usually uninvolved	May be swollen; possible blepharitis
Conjunctiva	May appear same in both types Often little objectively noted Sometimes edematous	May appear same in both types Papillary, but scattered follicles may appear Dry, no chemosis
Cornea	Generally not involved	May have superficial punctate keratitis involving inferior portion of cornea
Epithelial scrapings	Eosinophils almost always found sooner or later	No eosinophils
Cultures	Normal flora	Toxin-producing staphylococci found, or normal flora
Intracutaneous Staphylococcus toxin or toxoid	Moderate delayed response	Marked delayed response, at least 3 × 3 cm., usually more; may have marked immediate response also; may get systemic reactions (arthritis)
Response to vasoconstrictors	Local use often affords considerable symptomatic relief	Generally no great relief
Response to antihistaminics	Oral use often affords marked symptomatic relief	Generally no relief at all
Response to cortisone	Local use usually affords marked symptomatic relief	Relief in some cases, but not the rule
Best treatment	Elimination of cause	Staphylococcus toxin or toxoid injections

The characteristics of microbiallergic conjunctivitis due to the Staphylococcus and the differentiation of this entity are described in table I.

Fungi

Allergic conjunctivitis due to mold spores is not uncommon. Essentially an inhalant form of allergy, like pollen al-

lergy, the allergic reactions these fungi arouse have many of the characteristics of the immediate atopic response. Alternaria and Cladosporium, as well as yeasts, have been responsible.

Other types of fungi, of more pathogenic character, cause allergy only after a primary infective focus has been established. Here the mechanism is of the delayed tuberculin type. The dermatophytes, such as Trichophyton and Candida albicans (Monilia) are the chief fungi causing skin diseases. Thus, after a demonstrably infected primary dermatitis has occurred, *id* reactions may appear elsewhere. These *id* lesions reveal no fungi and disappear spontaneously when the original lesion is successfully treated. In the eye, such allergic reactions occur mainly as eczematous lesions affecting the eyelids; only rarely is the conjunctiva involved.

Helminths

Conjunctival allergy to intestinal parasites has been produced experimentally. Laboratory workers in this field, especially those dissecting the parasites, may contact allergic conjunctivitis from finger-to-eye contact. Reactions similar to hay fever, with conjunctivitis and asthma, also occur. Slaughterhouse workers may develop similar symptoms, which disappear when such exposure no longer occurs. At least two instances of recurrent allergic conjunctivitis related to Oxyuris infestation have been recorded.[5,9]

MICROBIALLERGIC REACTIONS OF THE EYELIDS

Edema of the Eyelids

While urticarial reactions of the eyelids have been ascribed to bacterial allergy, such a relationship has not been clearly proved. A more definite microbiallergic basis for such edema is seen in trypanosomiasis, in infestations with both round and flat worms, including Ascaria, Oxyuris, Trichinella, and in ancylostomiasis.

Infectious Eczematoid Dermatitis

Eczematoid dermatitis of the eyelids may arise on an allergic basis as a complication of bacterial, fungal or other microorganismal infection present elsewhere.[15] This basically allergic response to infectants must not be confused with the numerous types of dermatitis resulting from direct invasion of the skin of the eyelids by many bacteria, viruses and fungi. The latter are entirely distinct and not germane to the subject. Allergic reactions of the eyelids due to bacteria generally are the result of neighboring infection of the adnexae, although those due to rare syphilids and tuberculids need not follow this pattern. In fungal infections, however, the original focus may be far from the eye and entirely unrelated to it. In either event, the eczema appears to be, in larger measure, a manifestation of microbial allergy. While microbial dermatitis elsewhere on the body may result from a variety of infectants, including staphylococci, streptococci, and E. coli, as far as the eyelids are concerned the important bacterium involved is the staphylococcus. On rare occasions streptococci are responsible. Eyelid eczema due to fungus sensitivity, an unusual occurrence to begin with, appears mainly as trichophytid or moniliid. People with all types of infectious eczematoid reactions appear prone to develop secondary contact-type allergies, especially from medicaments. Since the reverse also happens—that is, infection secondary to primary allergic dermatitis — diagnostic problems may arise. About the eyelids, however, fewer such differential difficulties should occur, because the only type of infectious ecze-

matoid reaction of practical importance encountered — staphylococcal eczema — generally is associated with other ocular findings that afford valuable diagnostic clues.

Infectious eczematoid staphylococcal dermatitis, as differentiated from an allergic dermatoconjunctivitis and eczematous dermatitis, is evidenced by (1) blepharitis, often ulcerative; (2) meibomitis; (3) superficial epithelial keratitis involving the lower half of the cornea, seen on slit lamp examination; (4) strongly positive conjunctival and lid margin cultures showing many toxin-producing staphylococci, often numerically entirely out of proportion to the objective clinical findings; and (5) absence of eosinophils in epithelial scrapings.

Lid Margin Affections

In long-standing blepharitis, meibomitis, recurrent hordeolum and recurrent chalazion, all conditions in which staphylococcal infection plays an important role, secondary microbial allergy often supervenes. Desensitization therapy is an integral and often necessary part of the long-term treatment.

MICROBIALLERGIC REACTIONS OF THE CORNEA

A large part of corneal disease is allergic in nature.[10] Some of the earliest experimental work in general allergy as well as in ocular allergy was performed using the cornea as a shock organ. As early as 1909, Weekers[20] produced phlyctenules in rabbits sensitized to bovine tuberculosis. During the following twenty years, this work was confirmed and expanded by many investigators. Wessely,[22] in 1911, was a pioneer in demonstrating that the cornea is capable of reacting anaphylactically to produce parenchymatous keratitis.

The particularly great importance of allergy to microbial products in the production of corneal inflammation becomes apparent from table II. Some of the most common forms of keratitis are believed to arise on such a basis. Allergic reactions of the cornea to products of bacteria, protozoa, fungi, helminths and other similar products give a wide variety of corneal lesions. It is interesting that certain bacteria are associated with certain specific corneal changes. Thus, the classical picture of interstitial keratitis is caused by the spirochete of syphilis, and phlyctenular keratoconjunctivitis is most commonly traceable to the products of the tubercle bacillus. Similarly, marginal ulcers and infiltrates are characteristic of staphylococcal reaction.

TABLE II

MICROBIALLERGIC KERATITIS

1.	Lues, tubercle bacilli (other bacteria): interstitial keratitis
2.	Tubercle bacilli, staphylococci, other bacteria, Coccidioides immitis, nematodes: phlyctenular ophthalmia
3.	Staphylococci, bacillary dysentery: marginal infiltrates and ring ulcers
4.	Virus of herpes simplex: disciform edema of the corneal stroma

An allergic mechanism would appear to offer the best explanation for the occurrence of the two major types of interstitial keratitis encountered clinically, namely, luetic and tuberculous. Although largely a disease of a previous era, as already mentioned, the basic investigations linking this entity to allergy were among the first experiments in ocular allergy. Evidence for an allergic etiology is based on (1) the ineffectiveness of specific (antiluetic) therapy; (2) the effectiveness of corticosteroids and foreign protein therapy; (3) the rare onset

of interstitial keratitis during the course of systemic arsenic therapy (Jarisch-Herxheimer); and (4) experimental production of lesions identical in essential details to syphilitic parenchymatous keratitis.

Phlyctenular Keratoconjunctivitis

It is now generally accepted that phlyctenular keratoconjunctivitis is a manifestation of endogenous allergy not only to tuberculoprotein,[13,19] but also to other bacterial antigens, especially staphylococcal, and rarely to gonococcal and diplobacillary products. Instances have been encountered in patients allergic to coccidioidin, and in patients with leishmaniasis, trypanosomiasis, moniliasis, ascariasis, and helminthiasis. Phlyctenules have been produced experimentally by many allergy-inducing methods, using various antigens in appropriately sensitized animals. These include tuberculin, staphylococcal products, horse serum, and chemicals, which were instilled into the conjunctival sac. The dramatic response to corticosteroid therapy in all types of phlyctenulosis is further evidence suggestive of the allergic nature of the disease.

Marginal Ulcers and Infiltrates

Simple marginal or catarrhal ulcers and infiltrates are probably the most common manifestations of corneal allergy, usually of a microbiallergic character. While it has not been proved conclusively that marginal ulcers in bacterial conjunctivitis are allergic in nature, the weight of evidence favors an allergic etiology, especially as regards the Staphylococcus; here the antigen is probably the exotoxin of the bacterium. Marginal ulcers caused by other bacteria, such as the diplobacillus or Hemophilus conjunctivitidis (Koch-Weeks), may occur as a result of direct toxic action. In favor of an allergic etiology for such infiltrates and ulcers are the following findings:

1. Marginal ulcers rarely appear early in the course of staphylococcic or diplobacillary conjunctivitis. They occur commonly after the infection has been present for a period of time, often after repeated exacerbations.

2. Patients often show evidence of hypersensitivity to bacterial exotoxins in positive skin tests, or may have foci of infection (antigenic foci) in sinuses and tonsils and other remote areas of the body. However, in some cases the skin tests are only slightly positive.

3. It is difficult to isolate bacteria from scrapings of marginal ulcers.

4. Thygeson[18] claims that he has never observed a catarrhal ulcer caused by the pneumococcus. This is probably due to the fact that no exotoxin has been demonstrated in association with pneumococci.

5. There are many similarities between ulcers and phlyctenules, which are universally conceded to be allergic manifestations.

6. Marginal ulcers have been observed in atopic and contact allergies, such as tetracaine sensitivity, as well as in vernal catarrh.

7. Marginal ulcers in general respond very well to corticosteroids.

Ring Ulcers

Ring ulcers give the appearance of coalescing marginal ulcers but seem to have a different etiology, although they are allergic in nature. Systemic microbial allergy seems to play a role. We have encountered this condition in patients with ulcerative colitis, and Thyge-

son[18] has described it in bacillary dysentery.

Disciform Edema of the Corneal Stroma

Edematous reactions of the cornea of disciform character may arise in the course of various types of keratitis, particularly herpetic, or else may appear without any clearly demonstrable cause. The tendency has arisen to group all such instances of corneal edema under the term *disciform keratitis*. The disadvantage of considering all disciform stromal edema as one entity lies in the fact that whereas in herpetic keratitis corticosteroids are strongly contraindicated, in other disciform lesions such therapy may be of value. Beneficial effects in these cases may be due to a possible allergic component.

Originally, disciform keratitis was meant to describe a disk-like opacity situated in the corneal stroma, which often assumed twice its usual thickness in the involved area, and was associated with "descemetitis." Usually the corneal epithelium was relatively intact, and staining with fluorescein revealed little or nothing. Most such cases were attributed to herpes simplex infection on the basis of exposure to viral infections (colds, herpes labialis), often in association with trauma, and it was hypothesized that the virus had somehow entered the stroma through the epithelium. Many times the so-called point of entrance could not be demonstrated. Such cases were of themselves very serious; resultant central opacities, after the edema of the cornea had subsided, would reduce vision to the extent of industrial blindness. There seems no clear-cut evidence, other than circumstantial, that the condition is always due to herpes simplex. Sometimes herpes zoster and vaccinia seem causative. In still other instances the cause is not demonstrable.

The role of hypersensitivity in the causation of disciform keratitis has been accepted by many, although the evidence is partially hypothetical and inferential. Braley[1] has long been an important exponent of the allergic nature of disciform keratitis.

Recently Jones[6] has restated Braley's original concept in a graphic manner. He visualizes the corneal stroma as an immunological blotter which absorbs antigens arising from virus proliferating in the epithelium. The subsequent arrival of antibody gives rise to a fixed tissue antigen-antibody union resulting in edema or even necrosis.

It is my opinion that there are two types of disciform keratitis: (1) the type without clinical evidence of herpetic keratitis; and (2) the type occurring in association with demonstrable herpetic ulceration. It is hypothesized that in the first (classical) variety the cause has not been demonstrated and may possibly be allergic in nature; in the second form, where a herpetic ulcer pre-exists, while allergy possibly operates in the production of the stromal edema, the infectious component of the process predominates to the extent that corticosteroids must not be used.

MICROBIALLERGIC REACTIONS OF THE SCLERA

The widespread use of anti-infective agents and the control of syphilis and tuberculosis have resulted in the lessening importance of infection in the causation of scleritis. Thus while scleritis in itself is a relatively infrequent disease, the percentage of scleral inflammations of probable allergic origin is very high —perhaps higher than in any other ocular structure. However, because the sclera, unlike the conjunctiva, cornea and uvea, does not lend itself so readily

to the experimental investigation of allergic phenomena, it has been largely ignored in this respect. We are, therefore, forced to depend chiefly on clinical observations for the evaluation of the various allergic mechanisms that appear to operate in the production of scleritis. These indicate that many instances of scleritis are best explained on the basis of microbial allergy. In fact, if we consider certain of the collagen diseases, such as rheumatoid arthritis, to be due to bacterial (streptococcal) allergy, then it would be safe to say that most scleritis has a microbiallergic origin. Clinical and confirmative laboratory experiences indicate that such allergy is, in general, evoked chiefly by three organisms: Streptococcus, Staphylococcus, and the tubercle bacillus. However, the well known relationship between scleritis and what was originally termed the focus of infection, but which I prefer to call an antigenic focus, indicates that while these organisms dominate the picture, many others, such as enteric bacilli and perhaps even parasites, may be incriminated, although the evidence here is less clear-cut.

Table III classifies allergic reactions encountered in the sclera. The importance of microbial allergy is clearly seen.

TABLE III

ALLERGY OF THE SCLERA

1. Atopic allergy (immediate reaction):
a. Inhalants: episcleritis
b. Food allergy: episcleritis
2. Microbial allergy (delayed reaction):
a. Streptococcus, Staphylococcus: episcleritis, scleritis
b. Tubercle bacillus: scleritis, sclerosing keratitis
c. Other microorganisms: episcleritis, scleritis
3. Collagen diseases (Mechanism?):
a. Gout, rheumatic fever, rheumatoid arthritis, periarteritis nodosa: scleritis, episcleritis

Tenonitis

Except for the rare instance of tenonitis arising from direct infection, such as that complicating trauma or extraocular muscle surgery, the obscure etiology of tenonitis appears best explained in certain instances on the basis of allergy.

The rarity of the disease makes etiologic evaluation difficult. Its exudative character, its transient, recurrent, yet benign course, its association with collagen disease, its relationship and similarity to scleritis, and its response to corticosteroids and foreign protein therapy, all suggest that an allergic component may play a role in its causation. However, we know of no known instance where tenonitis has arisen on the basis of atopy such as food allergy. That microbial allergy may be in operation is suggested by its association with rheumatoid arthritis and infective antigenic foci. In this regard it is possible that the chemosis and other ocular findings in trichiniasis are due to tenonitis arising secondary to muscle infestation, either as toxic or allergic response to products of the trichinella.

MICROBIALLERGIC REACTIONS OF THE UVEA

There is considerable evidence, based upon anatomic, experimental, and clinical data, that allergy plays an important role in the causation of endogenous uveitis.[4] Anatomically the extremely vascular uvea offers a particularly favorable site for the concentration of proteins, antigens, and antibodies originating elsewhere in the body. Furthermore, it is known that local antibody formation can and does occur in the uvea itself. Since the uvea thus affords such an ideal shock organ for the study of allergic reactions, it is therefore not surprising that investigators in the field of immunology have utilized this structure experimentally in the production of allergic reactions

ever since the introduction of the concept of anaphylaxis. As a result, a great deal of the basic knowledge concerning hypersensitivity in general stems from such experiments. Allergic uveitis has been induced in laboratory animals by means of horse, cat, and other foreign sera, egg albumen, various microbial products, and by tissue proteins, either alone or in combination with Freund's adjuvants (heat-killed tubercle bacilli in mineral oil).

These observations show that all forms of allergic mechanisms operate in the production of uveal inflammation. Recent work by Silverstein and Zimmerman[12] again confirms this, indicating that endophthalmitis may be produced in the guinea pig by two distinct immunologic mechanisms: (1) delayed hypersensitivity and (2) antigen interaction with circulating antibody. By varying the method of sensitization, the route of administration and the dose of antigen, the several immunological mechanisms may be stimulated to produce ocular lesions. According to these authors, not only may acute nongranulomatous uveitis due to a delayed hypersensitivity result from microbiallergic processes involving bacterial antigens, but it may also arise as a consequence of tuberculin-type hypersensitivity to simple bland protein antigens such as crystalline egg albumin and bovine serum albumin.

The classification given in table IV is useful for the understanding of the clinical manifestations of uveal hypersensitivity.

Microbiallergic reactions of the uvea may occur in both nongranulomatous and granulomatous uveitis. In the anterior nongranulomatous variety, where no direct bacterial infection can be demonstrated, allergy appears to be the primary factor. Even in those granulomatous forms of uveitis where microorganisms can be demonstrated, a secondary microbial allergy often alters and aggravates the inflammatory state.

TABLE IV

ALLERGY OF THE UVEA

1. Anaphylactic and atopic reactions: Uveitis from sera, pollens, animal proteins, foods, drugs, angioneurotic edema
2. Microbiallergic reactions:
a. Nongranulomatous uveitis (allergy apparently the main factor): Streptococcus, tubercle bacillus, Staphylococcus, Gonococcus, Pneumococcus
b. Granulomatous uveitis (allergy not primary factor but may alter the nature of the inflammatory response): tuberculosis, syphilis, toxoplasmosis, brucellosis
3. Autogenous allergic reactions:
a. Sympathetic ophthalmia (allergy to autogenous uveal pigment)
b. Endophthalmitis phacoanaphylactica (allergy to autogenous lens protein)

While specific hypersensitivity to any of the bacteria listed in the table may cause anterior nongranulomatous uveitis, allergy to the streptococci is most commonly implicated. However, recent studies have failed to establish a correlation between elevated antistreptolysin-O titers and uveitis. Furthermore, skin tests with bacterial vaccines may be nonspecific, and convincing proof of the value of desensitization in a disease as variable and unpredictable as uveitis is not yet at hand.

All types of tuberculous uveitis have allergic components. Anterior uveitis may occur as a manifestation of tubercle bacillus hypersensitivity in the absence of direct infection, just as it is known to occur in phlyctenular keratoconjunctivitis and scleritis. Even in actual tuberculous infection of the uvea, as in posterior granulomatous uveitis, a secondary allergic reaction may dominate the clinical picture. Exacerbations of uveitis following testing, or therapy, with tuberculin are known clinically. Moreover, it

has been found that destruction of bacilli by means of chemotherapy can occasionally liberate sufficient antigen to cause a focal allergic response.

In ocular toxoplasmosis, both local and systemic sensitization to the toxoplasma occurs at the time that the organism forms pseudocysts in the retina. Generalized sensitization is manifested by a positive dye titer. When, years later, pseudocysts rupture, the hypersensitive tissues react with the liberated organism to produce an allergic reaction clinically manifested as a posterior uveitis.

Long before the concept of autoallergy was utilized to explain certain systemic diseases, the ophthalmologist had recognized that two unique types of autogenous allergy occur in the eye, namely, phacoanaphylactic uveitis and sympathetic ophthalmia. The concept of autoallergy, or allergy of the host to his own tissues, although still largely an experimental one, has other provocative applications in the field of uveitis. One may infer that the mechanisms of uveal allergy are even more complex than previously presumed, and that there are probably many avenues in which hypersensitivity mechanisms operate in uveitis. Just as autoallergy may some day serve to explain many now obscure uveal reactions, the hypothesis of autogenous hypersensitivity resulting from a microbial antigen may clarify much that is lacking for our full acceptance and understanding of microbiallergic uveitis.

MICROBIALLERGIC REACTIONS OF THE RETINA AND OPTIC NERVE

The optic nerve and retina are frequently the site of reactions resulting from exposure to various types of noxious agents. While most of such phenomena are obviously toxic in nature, often definite manifestations of allergy appear to occur. In general, allergic reactions of the retina and optic nerve appear to follow the usual lines of allergy elsewhere. With the exception of contact allergy, which obviously cannot occur, anaphylactic, atopic and microbiallergic reactions have been noted.

Toxic and hyper-reactive affections of the retina and optic nerve reveal little immediate objective evidence of tissue involvement, so that diagnosis depends on subjective findings, especially central vision and visual fields. In most instances of neuroretinal allergy, however, reactions are exudative and hemorrhagic, so that they are readily apparent on ophthalmoscopic examination. The only major exceptions would appear to be those rare cases of retrobulbar neuritis arising on an allergic basis.

Table V classifies all known instances of neuroretinal allergy.

TABLE V
NEURORETINAL ALLERGY

1. Anaphylactic and atopic reactions:	
a. Serum	
b. Food	
c. Pollen	
d. Drug	
e. Angioneurotic edema and urticaria	
2. Microbiallergic reactions:	
a. Tuberculin	
b. Other organisms	
3. Other reactions of possible allergic origin:	
a. Eye changes in periarteritis nodosa	
b. Central angiospastic retinopathy	

Experience indicates that most neuroretinal allergy is due to serum, foods, drugs, and pollens.[17(c)] Moreover, generalized angioneurotic edema and urticaria may likewise involve the optic nerve and retina. However, even though instances of microbiallergic neuroretin-

itis are rare, a number of rather convincing cases have been reported. Weizenblatt[21] has described such a reaction occurring on two occasions in a patient with marked cutaneous sensitivity to old tuberculin, five to seven days after skin testing with this antigen. Other reports[17(c)] describe similar exudative retinitis from tuberculin injections and in trypanosomiasis. Retrobulbar neuritis has resulted from prophylactic vaccination against rabies.

SUMMARY

Many of the important manifestations of ocular allergy appear to arise on the basis of hypersensitivity to products of bacteria and other microorganisms. While such microbiallergic reactions play a definite role in disease of the conjunctiva and eyelids, they are even more important in the cornea and in the uvea. Many common forms of keratitis are of microbiallergic origin. The etiologic significance of bacterial allergy in uveal disease is generally accepted; however, allergic mechanisms in uveitis may be of a more complex character than heretofore suspected. The little we know of scleritis indicates that microbiallergic reactions are of major importance. Even in the retina and optic nerve, allergic reactions to microbial products have been reported.

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DISCUSSION

ALSON E. BRALEY, M.D., Iowa City: Have you ever given a patient an intravenous injection of a few thousand killed typhoid bacilli and wondered what would happen? Most often they have a chill followed by a fever with joint pains, headache, and ache all over, really have the ague. The typhoid bacilli may even cause the eye condition to improve. Do you know what happened when you gave this injection? A lot of people have talked about it over the years and most of the evidence points to the fact that some substance in the microbe produces an unusual reaction in the body.

Microbes of all kinds release many antigenic substances. There may be some of the simple or complex nuclear substances present in the live or dead microorganisms themselves. These antigenic substances may be endotoxins which remain in the organism. Microorganisms sometimes secrete an exotoxin. Included in this group are several of the organisms which are well known to each of you. The staphylococci produce a variety of toxins, some of which are antigenic while others are only mildly antigenic. The staphylococcal exotoxin which produces food poisoning is only mildly antigenic to most people. I doubt if all of these so-called toxins secreted by bacteria have been isolated because I can visualize that an exotoxin can change as the nutrition of the microbe changes. A fourth possible antigenic substance is formed from the wall produced by the body to isolate microbes. These cells incompletely break down the microbe by-products and form new substances which in themselves are antigenic or toxic to the body.

Dr. Theodore has coined his new word "microbiallergic," which includes all the allergic reaction to microbes. He has presented many clinical descriptions of various eye structures that may illustrate "microbiallergy." These include the lids, the cornea and conjunctiva, the sclera, the uvea and the retina and the optic nerve. If a tissue can react unduly to a bacterial antigen then "microbiallergy" will result. Each of the eye structures react, some more violently than others, and some because of their physical nature react differently. The reaction of the tissue is our clinical evidence of allergy. Since most bacterial allergy is delayed, the differential diagnosis between the direct infection by the organism and the manifestation of allergy may present a very difficult problem. The bacterial laboratory may be of help here along with skin tests and observations of the type of lesion in the eye. If there is a direct infection, there are usually a large number of causative organisms present. If there is an allergic reaction, then the organisms usually are absent or very sparse.

Virus infection and herpes do not present a special problem. In the case of herpes simplex infection, hypersensitivity or allergy may not be a factor. When no allergy is present, the lesion clears spontaneously or in a very short time. When allergy is present, and it takes time for hypersensitivity to develop, several reactions occur. Disciform keratitis and the severe iritis that sometimes accompany herpes simplex keratitis are certainly allergic reactions. All the evidence points in this direction. I think that by the careful use of cortisone or the other anti-inflammatory agents, one can control the edema of the stroma and the iritis until the virus become latent again.

It has been a pleasure to read Dr. Theodore's paper. I highly recommend it to every practicing ophthalmologist who treats infections in and about the eye. I would especially recommend it to the ophthalmologist who treats patients with chronic conjunctivitis.

A NEW-TYPE SILICONE ROD FOR SCLERAL BUCKLING

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IN recent years, scleral shortening operations for retinal detachment have received general acceptance. Scleral resections, scleral outfolds, scleral undermining, and scleral buckling have all been utilized for reduction of ocular volume in retinal detachment surgery. The scleral buckling operation employing polyethylene tubing as described by Schepens[3] is currently the most commonly used scleroplasty operation in this country. Since the introduction of the encircling polyethylene tube operation for retinal detachment, two modifications pertinent to this presentation have been advanced and are generally accepted. The first of these modifications is the injection of an antibiotic into the lumen of the polyethylene tube at completion of the operation; the second is the use of a polyethylene sleeve over the junction of the ends of the tube. It may be assumed that these modifications resulted from complications arising in the original encircling polyethylene tube operation. Lincoff[2] described several cases of chronic granuloma at the site of the suture tie and tube junction and believes that this complication can be prevented by utilizing these modifications.

The authors of this paper have encountered similar granulomata using encircling silicone rubber tubing and a silk suture. The initial use of silicone tubing was undertaken several years ago in an effort to avoid these chronic inflammatory reactions. Animal experiments, to be reported separately, led us to believe that the granulomatous reactions were the result of the dead space in the lumen, a tube containing a silk suture, and tissue fluids, which create a culture area for bacterial growth.

One of the authors (W. G. E.) observed over a period of several years that solid silicone rods embedded in segmental areas of the sclera have not produced inflammatory granulomas. This procedure was utilized following the report of Girard,[1] who suggested the use of an encircling solid silicone rod. However, the overlapping juncture of the ends of the encircling silicone rod was found cumbersome and bulky. Since no chronic inflammations were encountered with the encircling silicone rods, study of this procedure seemed indicated.

The following method was devised for creating a scleral buckle in which there was no luminal dead space such as is created by the conventional use of an encircling polyethylene tube. A silicone

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This study was supported by Public Health Grant B2481. The silicone rod (X-30146) was furnished and extruded by the Dow Corning Center for Aid to Medical Research, Midland, Michigan. The suture materials were furnished by Ethicon, Inc., Somerville, N. J.

Presented as a *New Instrument* at the Sixty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1960, Chicago.

rod extruded with a 3-0 mersilene core or 3-0 braided stainless steel wire was used. This rod is buried in a scleral lamellar undermine as large as is necessary to apply adequate choroidal diathermy, depending on the characteristics of the retinal detachment, as is done in conventional scleral buckling (fig. 1, A). placed around the rod near the knot (fig. 1, E). The area is carefully irrigated and cleaned with physiologic saline solution and is dried with cotton sponges. Silicone glue may then be applied about the knotted suture and rod (fig. 1, F). This silicone glue creates a permanent seal of the rod ends. The silicone sleeve

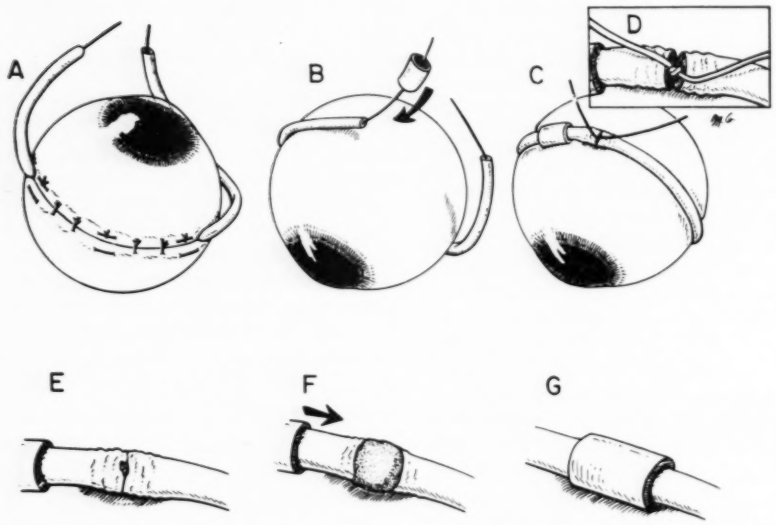

FIG. 1—Illustration of the use of a new type of silicone rod and suture in scleral buckling.

The rod encircles the undissected equatorial circumference of the globe over the surface of the sclera. The silicone rubber is stripped off the suture core, allowing about 1/32 of an inch overlap of each end (fig. 1, B). The rubber on the suture ends is then pushed back. When the encircling suture is tied, the slight excess rubber crowds or butts the knot, making a close approximation of the juncture (fig. 1, C and D). A small sleeve of silicone rubber was previously is then advanced around the glued juncture to create a permanently watertight joint (fig. 1, G).

Silicone rubber has the following advantages: (1) it may be autoclaved; (2) it does not deteriorate with age; (3) it is adhesive to all other materials or fluids; and (4) it is inert to body tissues.

As in all ocular surgery, meticulous attention must be paid to a sterile field

and the prevention of contamination regardless of any modification of operative approach.

SUMMARY

The scleral buckling operation has been widely accepted as a means of treating retinal detachment. Occasional inflammatory granulomas following scleral encircling operations with polyethylene and silk suture have been encountered. Clinical experience with silicone rubber rods and the suggestion that the operation using an encircling polyethylene tube with luminal-silk may cause chronic granuloma led to the development of a method for treating retinal detachment which utilizes an extruded silicone rod with suture core* and a fused, watertight silicone juncture in an effort to avert the occasional chronic granuloma produced by the encircling scleral buckling operations for retinal detachment.

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*Available from Storz Instrument Company, St. Louis.

THE TRABECULOTOME

An Instrument for Trabeculotomy ab externo

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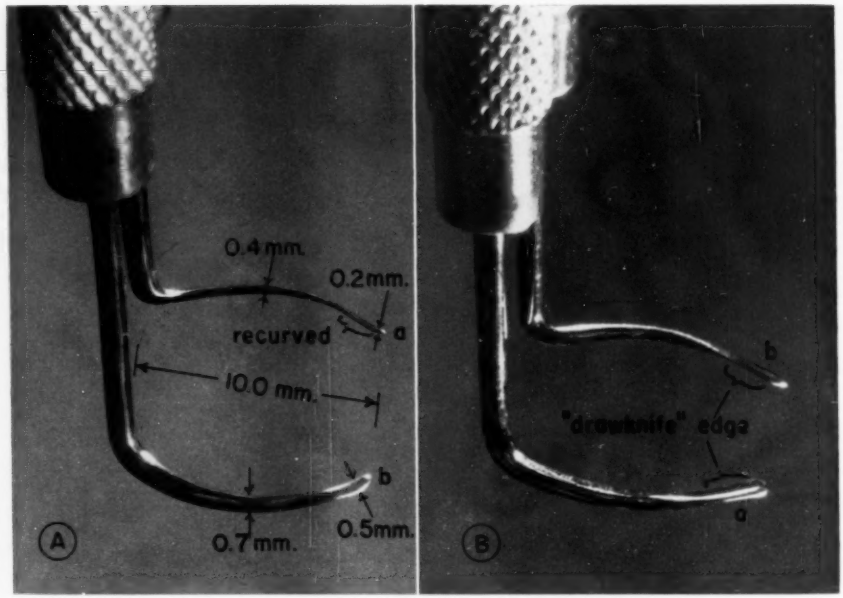

FIG. 1—Two models of the trabeculotome: (A) blunt model; (B) model with "drawknife" edge. The handles are positioned closely so that the second handle is hidden. The trabeculotomes come in pairs which consist of an instrument for right thrust and another for left thrust.

THE instrument presented here has been designed for the purpose of performing a trabeculotomy ab externo.[1,2] The essence of this operation, devised primarily for developmental glaucoma, consists of introducing an appropriately shaped spatula into the scleral groove after making a radial incision through the external limbus and sclera. On correct withdrawal, the spatula will sever the trabecular meshwork, exposing the canal of Schlemm if one is present, or at least some part of the innermost layers of the meshwork.

The instrument itself consists of a spatula or blade fastened to a round Kirby-type knurled handle. The spatula is shaped to follow the curve of the limbus, except for about 2.5 mm. at the

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Presented as a *New Instrument* at the Sixty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1960, Chicago.

tip, where it is recurved, the gross radius of its curve being 6 to 6.5 mm. The linear distance from handle to tip is about 1 cm. The oval cross-section of the spatula is tilted at about 45 degrees from the plane of the long curve. The spatula tapers gently from base to tip; it is 0.7 mm. wide and 0.4 mm. thick at midpoint, and 0.5 mm. wide and 0.2 mm. thick in the recurved portion of the tip.

One model (fig. 1, A) is blunt, for sectioning the meshwork by gentle pressure upon withdrawal. The other (fig. 1, B) has a "drawknife" edge which starts 2.5 mm. in back of the tip and extends from 2 mm. to within 0.5 mm. of the tip. This knife edge is level with the uppermost part of the oval cross section so that it will cut well ahead of the scleral spur as the spatula is withdrawn. Both the blunt and the sharp trabeculotome come in pairs—each pair consisting of an instrument for thrust to the right and another for thrust to the left.

The results obtained with this instrument in experimental eye surgery and in surgery on living eyes will be published elsewhere.[1,2] Slides showing the preoperative and postoperative chamber angle drawings and histologic specimens were shown, but are not reproduced in this note.

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PROSTHETIC ARTICULATIONS IN STAPES SURGERY

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IN the revived era of stapes mobilization, the first group of approaches following Rosen's leadership[17-19] involved indirect force applications transmitted to the ankylosed area to liberate the fixed stapes from its pathologic attachments and remobilize it; thus force was applied either to the incus, to the incudostapedial articulation, to the neck, or to the crura, utilizing various shaped picks and blunt instruments. In these early attempts, many successful results were obtained. This indirect type of operation is still in use throughout the world, with a yield of approximately 30 per cent successful mobilizations. In the other 70 per cent of cases, the indirect approach usually resulted in either the disruption of the incudostapedial joint or crural fracture, so that the force application never quite reached the crucial ankylosed area and remobilization was not achieved.

As experience in this approach accumulated, it became obvious that it was not desirable to sacrifice the ossicular chain for the advantage of a 30 per cent success group, and that more precise methods directed to the footplate region itself might increase the number of good results and make the operation more desirable. The locus of force application began to leave the incus, the stapedial neck, and the crura, and began to approach the footplate region. Originally, the first efforts in this new direction were towards force through the otosclerotic focus in the anterior or posterior peristapedial (peribasal or pericrural) region. Picks, chisels, and gouges, either mallet or motor-driven, were used in this direct attack. Heermann[4,5] of Essen was the pioneer in advocating delicate, precise chisel techniques. Later, the operations of anterior crurotomy and then partial anterior crurectomy were developed, which resulted in monopod posterior mobilizations.

Peribasal translesion fracture is still very much in use, and will probably find a limited place in the future plan for orderly stapes surgery. There is no doubt that in many patients the footplate can be successfully and permanently remobilized by direct fracture through the footplate region. There is much evidence that it is possible to obtain a permanently mobile footplate even though the fracture has gone directly through an otosclerotic focus. However, it appears that more successful long-range results can be obtained if the fracture area does not go directly through the otosclerotic process, but through the margin between the otosclerotic lesion and the normal footplate. A sufficient number of excellent results over long-range periods exists in this latter group to justify its retention as a valid technique (under special indications only), to be utilized with a high degree of safety in the surgery for stapedial ankylosis.

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With the redirection of effort to the footplate region, the number of successful results doubled and the 30 per cent "successful" results of the "indirect" mobilization era jumped to at least 60 per cent in the "direct" era. This increase was the most important factor in the replacement of the fenestration operation by the stapes operation as the primary treatment of otosclerotic deafness. With the emergence of the direct footplate era (1957-58), the fenestration operation definitely became secondary in the treatment of otosclerosis and many of the fenestration surgeons who had scoffed at stapes surgery and had predicted its early demise were now "jumping on the band wagon" and pointing with pride to their stapes results. Some of the more experienced and skillful surgeons reached "successful" primary results in 80 to 85 per cent of cases.

However, when some of the excellent primary results began to diminish by virtue of reankylosis (Bellucci and Wolff[1,2]) and when the reankylosis figures began to mount, it became obvious that some other approach would be necessary, particularly in the more extensive lesions. This invited the use of a more radical group of procedures involving partial or complete stapedectomy. It is interesting to note that we then came full circle in the surgery of otosclerosis, from the early days of Kessel[10,11] and his contemporaries who performed total stapedectomies. They were followed by Miot[14] and colleagues who performed mobilizations. This era, in turn, was replaced by that of the Lempert[13] fenestration operation, which ignored the oval window. We then returned to the oval window via the Rosen mobilization,[17-19] and now we have come completely around the circle back to stapedectomy as originally advocated by Kessel (fig. 1). The return to stapedectomy occurred spontaneously and almost simultaneously throughout the world. Its re-emergence was logical when realization of the extent of the reankylosis problem became widespread. It is difficult to trace any direct chronological relationship of the various new stapedectomy procedures as they evolved, so they will be described in anatomical order.

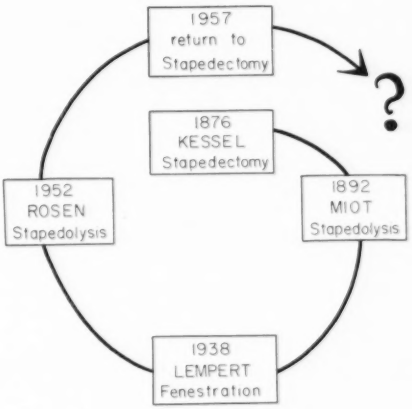

FIG. 1—From stapedectomy to stapedectomy to ?

A. STAPEDECTOMY: ITS VARIOUS FORMS

1. *Partial Anterior Crurectomy*

The excellent results reported by Fowler[3] and others who originally advocated anterior crurotomy were somewhat downgraded by reunion of the crural fragments. Accordingly, excision of part or most of the anterior crus was adopted by some surgeons (L. Holmgren[6]). Partial anterior crurectomy was quite effective when combined with footplate fracture to isolate the anterior lesion (fig. 2) and anterior one half of the footplate. The mobile posterior footplate segment, attached to the posterior crus, was now free to transmit acoustic energy to perilymph via a posterior monopod mobilization (fig. 3).

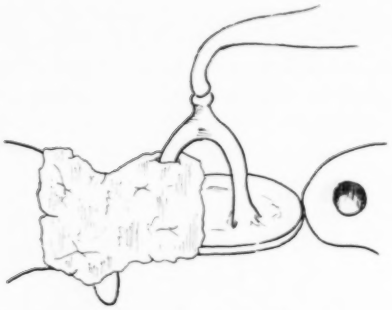

FIG. 2—Extensive anterior ankylosis with crural involvement.

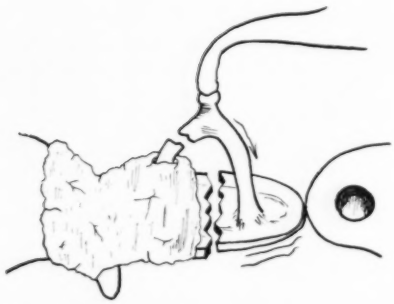

FIG. 3—Partial anterior crurectomy of posterior monopod basolysis.

In occasional cases, the ankylosing lesion is primarily posterior (fig. 4). In that event, a posterior partial crurectomy accompanied by footplate segment attached to anterior crus should result in anterior monopod mobilization (fig. 5).

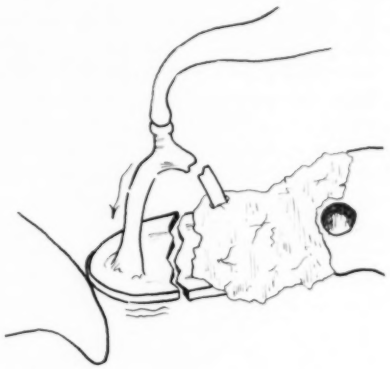

FIG. 5—Partial posterior crurectomy with anterior monopod basolysis.

2. *Anterior Crurectomy, Hemistapedectomy and Stapedioplasty*

Proceeding along the same basic anatomical-surgical principle of monopod mobilization, several otologists have advocated the procedures of anterior crurectomy and hemistapedectomy (Juers,[8,9] Hough[7]), in which they removed most of the anterior crus en bloc (fig. 6), fre-

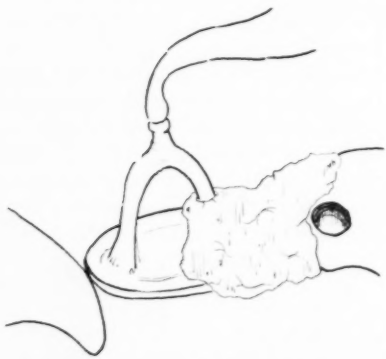

FIG. 4—Extensive posterior ankylosis with crural involvement.

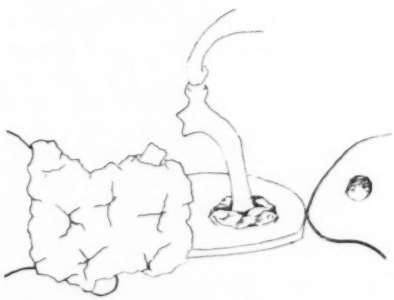

FIG. 6—Massive anterior lesion about to be removed following anterior crurectomy.

quently along with the anterior half of the stapedial footplate including a wedge of the otosclerotic anterior lesion (fig. 7). This then resulted in a simple mono-

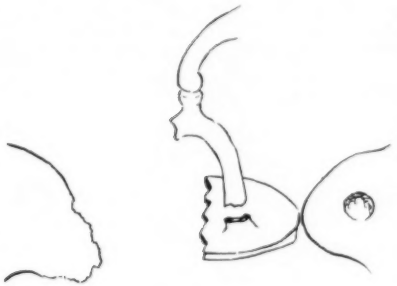

FIG. 7—Resection of footplate attachment of posterior crus prior to redirection in stapedioplasty.

pod mobilization without any change in the position of the posterior crus and the posterior stapedial segment.

Juers advises a redirection of the posterior crus in monopod fashion either to the central or to the anterior portion of the oval window (fig. 8). It may be ac-

FIG. 8—Redirected posterior crus on gelfoam cover over anterior vestibular opening.

companied by no oval window replacement or in some instances by replacement through vein graft, connective tissue, or gelfoam.

3. The Interposition Operation

Michel Portmann[15,16] described the interesting approach of interposition in which a "platinectomy" or "basectomy" or footplate excision is performed with preservation of head, neck and crura. The removal of the entire stapedial footplate is followed by replacement with a vein graft (fig. 9). This allows the pres-

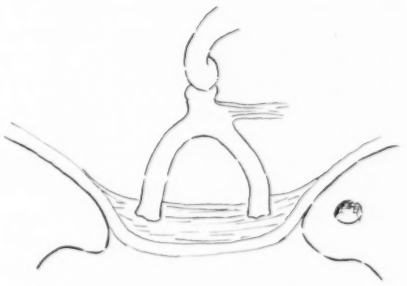

FIG. 9—Portmann interposition operation. Otosclerotic footplate has been removed and replaced with vein graft. Crura in contact with vein graft.

ervation of the stapedial tendon and maintenance of the integrity of the incudostapedial joint, but reattaches the medial aspects of the crura to a mobile vein graft rather than to a rigid footplate.

4. Subtotal Stapedectomy and Prosthetic Replacement

This is the most widely used version of stapedectomy and may well remain a major stapes procedure in the future. In this procedure, following section of the stapedial tendon and incudostapedial joint, the head, neck and crura are fractured away en masse and the medial crural insertions and entire footplate remain. A substitute stapes is created by the installation of a prosthesis between incus and footplate, the footplate remaining *in situ* but being mobilized by one of several methods. The two most

common stapedial replacements have been plastic (polyethylene) and metal (stainless steel or tantalum) struts. The strut problems will be described later in a special analysis.

Mobilization of the footplate *in situ* to allow transmission of energy from strut to vestibular perilymph can be accomplished in several ways.

The simplest method is *total footplate lysis,* which is possible in relatively mild types of ankylosis in which the anatomical integrity of the footplate circumference (perimeter) is still inviolate. This condition may exist either in completely virginal (uninvaded) or ossified (invaded) footplates. Even though the annular ligament has been deformed or destroyed, the anatomical configuration of the oval window may be intact and the footplate may be completely mobilized during the fracture maneuver of subtotal stapedectomy. In such cases, the medial end of the strut will simply press on the mobile footplate and transmit energy in the same manner as normal crura. The strut will require rather tight positioning to expedite efficient energy transfer (fig. 10).

FIG. 10—Total footplate lysis. Blunt end prosthesis in pressure contact with a mobile footplate.

A footplate fixed anteriorly and posteriorly with or without concomitant circumferential invasion will frequently demonstrate a completely normal central virgin island (fig. 11). Such islands

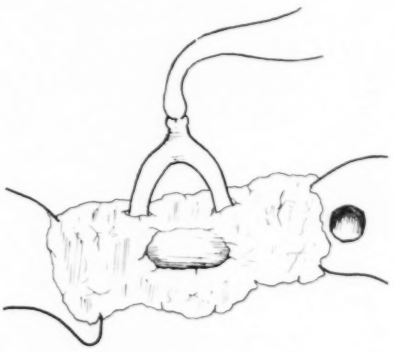

FIG. 11—Extensive total ankylosis with central footplate island.

have been observed repeatedly in patients with otosclerotic histories of even 30 and 40 years' duration. It is possible to treat such an island in several ways:

a. It may be mobilized en masse around its perimeter and the strut will be applied to it under slight tension in the same manner as the totally mobilized footplate (fig. 12).

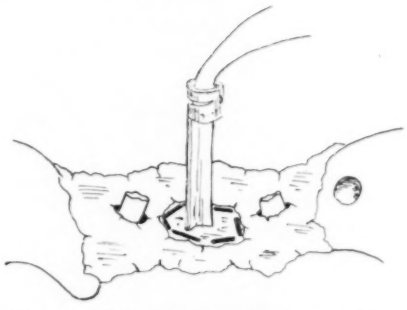

FIG. 12—Completely mobile virgin island acting as a "lid" in contact with a blunt prosthesis.

b. It may be fractured in a crucial manner and the medial end of the strut thrust through the fracture lines into the

vestibule. Such a strut will usually be notched medially (fig. 13).

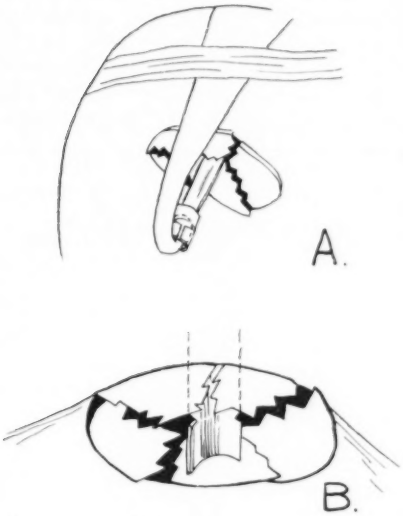

FIG. 13—(A) Notched prosthesis placed into the vestibule through fractured virgin island. (B) Vestibular view of notched prosthesis in contact with virgin island fragments.

c. It may be completely pulverized, and either covered with autogenous tissue or gelfoam or left uncovered. A strut will then be placed in contact with the pulverized island on its covering tissue.

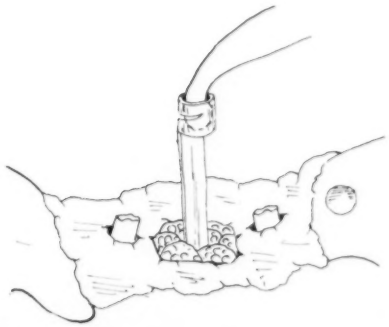

FIG. 14—Polyethylene articulated strut in place on fat graft covering vestibular opening.

d. The entire island may be removed, the resultant vestibular defect closed either with a vein or fat graft or gelfoam, and a contacting strut installed (figs. 14, 15 and 16).

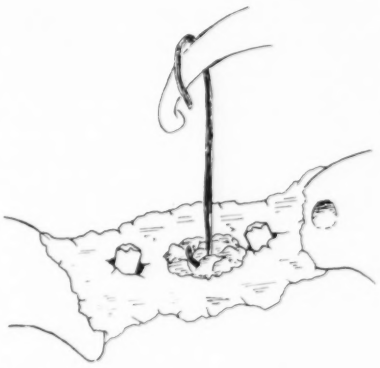

FIG. 15—Stainless steel strut in place over fat graft closing vestibular opening.

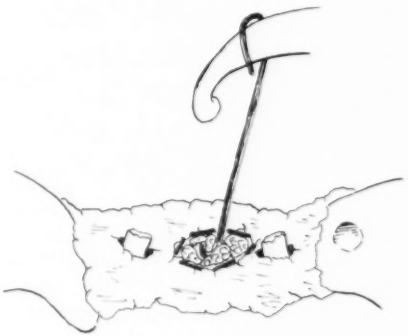

FIG. 16—Stainless steel strut in place over gelfoam-covered vestibular opening.

A footplate invaded by otosclerosis so that no virgin island remains either will become thickened but still retain anatomical boundaries or will become fused amorphously to the oval window margin so that no vestige of previous oval window frame can be recognized (fig. 17). In the latter case, otosclerotic in-

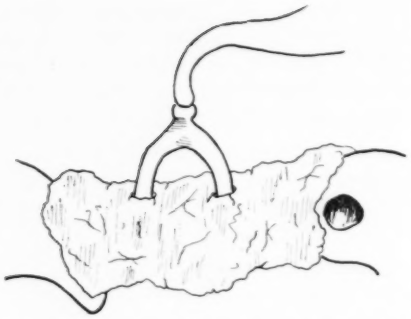

FIG. 17—Total footplate ankylosis: amorphous mass; no island.

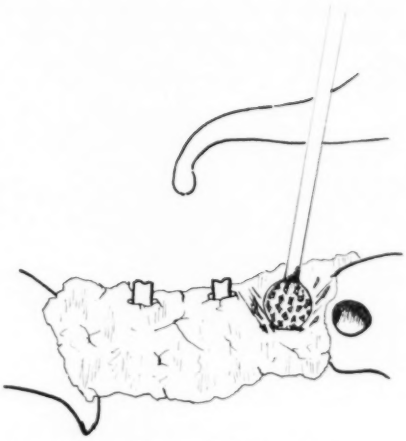

FIG. 18—Amorphous footplate; burr fenestration.

vasion may occur in any portion of the otic capsule, with frequent large otosclerotic exostoses presenting from the promontory or extending into the vestibule.

In the former variety (i.e., with normal boundaries) probably the best approach is total stapedectomy (to be described later). In amorphous diffuse otosclerosis in which oval window boundaries have been destroyed, it is questionable whether any oval window approach is desirable. However, the author and others have observed at least short-term successful results in many such cases treated by artificial fenestration of the amorphous mass with a burr (fig. 18) and combined with a prosthesis and a tissue or gelfoam seal. Where a normal oval window boundary still exists, excellent results (at least temporarily) have been obtained by pulverization techniques, chisel fractures with displacement of fragments and even intravestibular dislocation of a footplate too broad to allow for easy delivery. In all of these techniques, "success" (temporarily) must depend upon maintenance of the desired displacement and adequate prosthetic contact with a membrane-covered perilymph area.

5. *Total Stapedectomy*

In returning to the circle started in 1876 by Kessel (fig. 1), we see another example of "nothing new under the sun." However, these returns in human creativity are not always simple returns. New depth usually accompanies the returning concept, and this is certainly true in stapes surgery.

The essential feature of total stapedectomy is the total removal of the footplate. There is apparently little difference between the effectivity of acoustic energy, transmission between the intact superstructure (head, neck and crura) of a stapes (as in the "interposition" operation of Portmann) and an *articulated* prosthesis, with the exception of the unknown sequelae to the cochlea of deprivation of stapedius muscle protection effect in the prosthetic case. What is said of the *articulated prosthesis* is not necessarily true of all prosthetic struts. A loose sliding metal strut or a loose, partly attached polyethylene strut is certainly inferior to a naturally articulated stapedial "superstructure."

Total removal of footplate or oval window fenestration demands not only the utmost in microsurgical technique but a critical awareness of the problems inherent in exteriorization of the vestibule and the special demands placed upon the hydrodynamics of perilymph production and absorption. The latter problems first manifested themselves during the development of the Lempert fenestration operation. They are perhaps more acute in refenestration of the oval window, because anatomic relationships are more crucial.

That due consideration of these inherent dangers is lacking in radical footplate surgery is amply demonstrated by the alarming number of "dead labyrinths" reported recently. Unfortunately, most of these reports have not yet been published, and it is questionable if all of these cases will ever find their way into print. They have been reported verbally at every recent otologic meeting, especially in informal discussions. However, it seems likely that most of these problems are not insuperable and that measures to protect the endolymphatic labyrinth will be forthcoming.

It is regrettable that experimental methods to study these problems have, with few exceptions, lagged behind clinical investigation. It is to be hoped that increased laboratory facilities will expedite such fundamental studies.

Technical details of total stapedectomy will be described later. The basic principles, however, involve the following points:

a. Gentle decompression of the perilymph space rather than violent "uncorking"

b. Careful removal of footplate fragments and bone dust

c. Blood-free field

d. Very delicate suction

e. Adequate immediate sealing of oval window, preferably with a tissue transplant

f. Careful positioning of strut to ensure its integration with the newly created mobile membrane

Tissue transplants or free grafts have played the most important part in the development of radical oval window surgery. The vein graft as developed by Shea[21] has been in the forefront of graft techniques. Shea has had extensive experience with this technique and feels that it may replace most of the previously used procedures.

In Shea's technique, the head, neck and crura are removed in the usual manner and the entire footplate removed from the oval window frame. Prior to this removal, however, a segment of vein is removed from the dorsum of the hand under local anesthesia. A small thin-walled vein is preferred.

Excessive connective tissue is trimmed from the adventitial surface and the vein is split lengthwise. After careful trimming for size so that it will cover the oval window and portions of the fossa ovalis, it is kept in sterile Ringer's solution until needed.

Mucoperiosteum is removed from the margins of the oval window to create a raw surface. The footplate is removed delicately by fine angular dissectors and hooks. If necessary, a microdrill is used to facilitate removal.

As soon as the footplate is removed, the vein graft is placed in position over the window, adventitia in and intima out, and a polyethylene strut placed between it and the incus. A slight indentation by the strut is desirable (fig. 19).

A modification of the vein graft has been advocated by Kos.[12] He utilizes a stainless steel strut with a shepherd's crook over the long process of the incus.

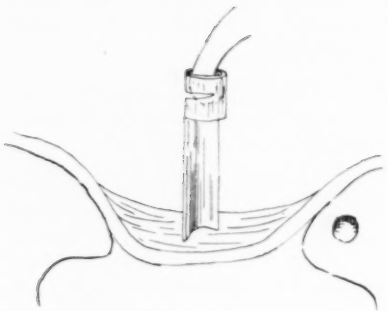

FIG. 19—Shea vein graft over oval window after footplate removal. Articulated polyethylene strut in position between incus and vein graft.

The medial aspect of the strut is tied around an unslit vein removed from the dorsum of the hand.

This vein plug is placed into the oval window to seal the vestibule and form another version of a pseudofootplate, with the steel strut transmitting energy from the drum-ossicular chain to the vestibule (fig. 20).

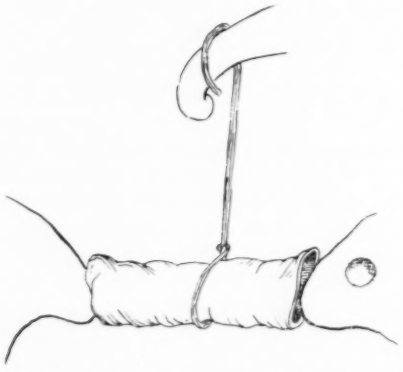

FIG. 20—Vein plug of Kos in position over oval window with stainless steel wire connection to incus.

Other free grafts utilized have been conjunctiva, mucosa, connective tissue and fat. The fat graft technique has been used extensively by Schuknecht,[20] and by the author with an articulated polyethylene prosthesis.

The fat graft used by Schuknecht utilizes a stainless steel strut tied to a piece of fat taken from the ear lobe. The fat graft plugs the oval window in a manner similar to the vein plug with the steel strut transmitting acoustic energy from the drum-ossicular chain (fig. 21).

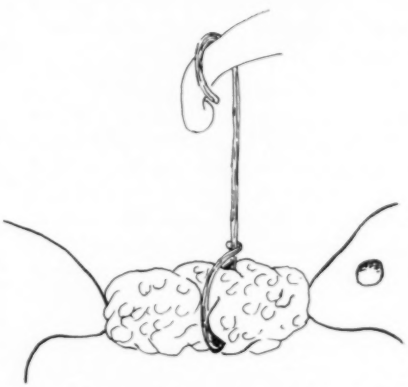

FIG. 21—Schuknecht fat graft in position over the oval window with stainless steel wire connection to incus.

Excellent results, as high as 90 per cent "successes," have been reported with vein grafts, vein plugs and fat grafts. Thus the tendency to adopt total stapedectomy is widely evident.

However, the final chapter on total stapedectomy cannot be written for many years. Ultimate fate of the tissue grafts, gelfoam and prostheses is unknown. The status of the cochlea on a long-range basis remains to be studied. Response of the newly opened and newly sealed vestibule to various types of infections of the upper respiratory tract remains unknown. The sequelae of deprivation of stapedius muscle protection remain to be evaluated.

All of these unknowns must be

weighed against the large, although still incomplete, battery of data concerning the Lempert fenestration operation. The indirect (semicircular canal) route cannot be ignored and must not be left out of the total plans for the surgical treatment of otosclerosis. The otologic surgeon planning reconstruction of the stapedial-vestibular articulation must be prepared to use both metal and plastic prostheses. With either type he must achieve a functioning articulation with both incus and footplate (or neo-footplate).

A. METAL PROSTHETICS AS STAPES SUBSTITUTES

The use of either stainless steel or tantalum wire (gauge 32) has been advocated by a number of surgeons, notably Schuknecht and Kos. A piece of wire is bent to form a shepherd's crook, which hooks around the long process of the incus laterally. The medial end of the metal strut (approximately 4 mm. long) is fashioned into a rounded base to transmit energy to the oval window region in one of several ways.

The lateral end is fashioned into a hook-like shepherd's crook to fit snugly around the long process and is crimped into position by the use of a modified alligator forceps. If the hook is too tightly clamped to the incus, ischemic necrosis will result. This should be avoided. A slight amount of sliding is permitted. The medial aspect is placed upon the footplate, following which the footplate is fractured slightly inwardly if it still has the integrity of a total mobile footplate. The slight pressure exerted by the strut in keeping the footplate pushed into the vestibule is usually sufficient to create rather good linkage so that transmission of acoustic energy is reasonably efficient. In the event that the footplate has been invaded by otosclerosis, it (or a virgin island) may be fractured loose and a single fragment or several fragments covered by small pledgets of gelfoam which have been saturated with Hydeltrasol (predisolone phosphate sodium) solution. The medial end of the strut is then placed on the mobile footplate region with moderate pressure. This will permit a partial footplate segment which is completely mobile to transmit energy from incus via strut to perilymph. In instances in which the footplate is totally invaded by otosclerosis, it may be removed either completely or partially and a fat graft or vein graft placed over the aperture. The strut is placed on the graft to transmit energy through the new, more compliant oval window membrane. The strut or "pin" carries with it certain potential disadvantages:

1. The lateral hook around the long process may become too loose and fail to transmit incus motion reliably.

2. A very tight hook may predispose to incus necrosis.

3. The ultimate effect of steel or tantalum upon the physicochemical and other properties of perilymph and endolymph is unknown.

In some cases, however, the metal pin or strut is the only possible solution to a stapes problem, particularly in instances in which there is necrosis of the lenticular or long process of the incus (fig. 22), making it impossible to articulate a polyethylene prosthesis adequately. In cases in which the oval window recess is so small that a polyethylene prosthesis would impinge upon either promontory or the facial canal, the pin becomes the only possible solution to transmission linkage from incus to oval window. In the event that only a small virgin island or drilled island is accessible to the vestibule, only a metal pin can be used for linkage in such cases.

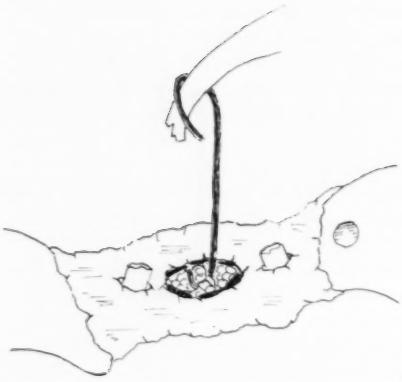

FIG. 22—Stainless steel strut is only transmission possible in instance of lenticular process necrosis. In this case it contacts the gelfoam-covered vestibular opening.

patients with less favorable anatomical circumstances this prosthesis was impractical, and it became necessary to substitute for the simple length of tubing, a segment of tubing the medial aspect of which had been diagonally cut

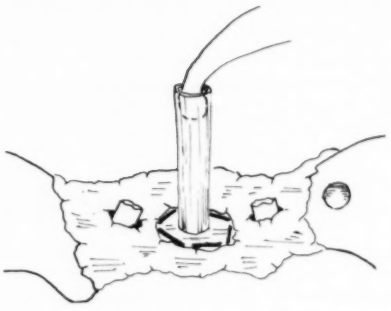

FIG. 23—Primitive form of polyethylene prosthesis. Simple length of tubing between incus and mobile virgin island.

B. POLYETHYLENE PROSTHESES

Early in the history of the new stapes era revived by Rosen, a number of surgeons, challenged by reankylosis and other problems, began to utilize stapedial replacements. The author, in 1956, performed a number of total stapedectomies and substituted polyethylene prostheses of varying types between incus and footplate. These early experiments were intentionally few in number so that long range observations of the fate of polyethylene might be available before it was advocated for more general use.

The anatomy of the polyethylene prosthesis has undergone quite an evolutionary development which can be traced as follows:

A simple segment of polyethylene tube was the earliest version of the prosthesis and served to connect the lenticular process to the central footplate area. In its simple form (fig. 23) this was a very adequate strut under circumstances which were anatomically ideal. Unfortunately, however, in the large group of so that it could enter the narrow fossa ovalis region without impinging upon the promontory (fig. 24). The diagonal strut prosthesis is still in wide use in most techniques, including the vein graft. Under most circumstances it is probably adequate. However, in many cases the bulge of the promontory either in its normal state or following otosclerotic infiltration makes it necessary to change the cut of the strut so that there is less opportunity for friction between strut and promontory. Because of promontory problems and situations where a narrow fossa ovalis made the diagonal strut inoperative, the medial aspect of the strut came to resemble more and more a true strut and less and less the segment of a cylinder (fig. 25).

As special problems arose requiring more delicate transmission channels (such as too small virgin islands surrounded by massive amorphous otosclerosis), the strut became narrower

until it occupied the width of about 1 mm. or less and represented about 90 degrees of the circumference of the cylindrical tube (fig. 26). Finally, in our usage the strut is virtually a straight piece of polyethylene, reserving the tubal aspect only for the lateral incus attachment region. There is no tube at all in the transmission strut itself (fig. 27).

Fortunately, the polyethylene strut has been shown to be relatively inert. Under proper aseptic conditions the intratympanic and intravestibular insertion of such a strut is a safe procedure.

Other problems in the use of the strut soon became apparent, however; the attachment of strut to incus remained a problem which is now approaching solution. Examination of incus-strut unions

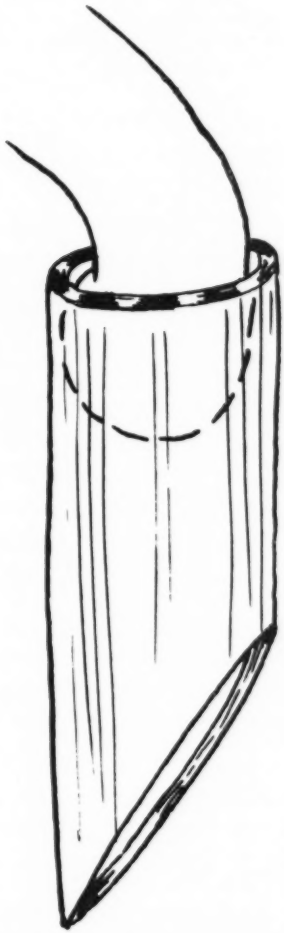

FIG. 24—Standard polyethylene strut with diagonal cut.

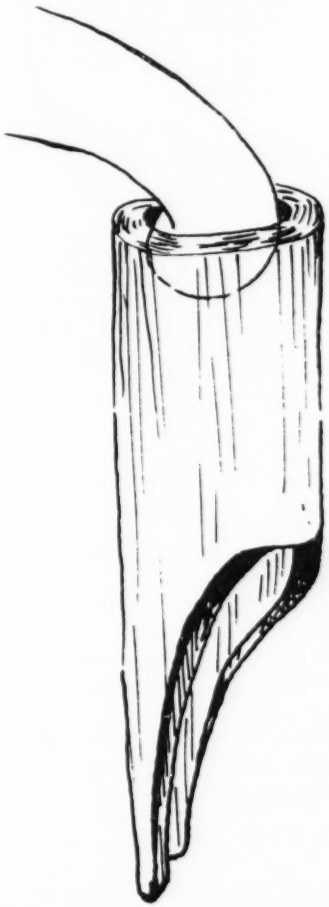

FIG. 25—Modification of polyethylene strut to avoid frictional contact with promontory.

FIG. 26—Further modification of medial aspect of polyethylene strut to fit a narrow oval window.

at revisions revealed that in the vast majority of cases a fibrous tent had grown around the union so that the incus-strut union was virtually a fixed and articulated joint (fig. 28) capable of push-pull transmission, a definite acoustic requirement of the normal incudostapedial joint. However, in some cases at revision it was found that no such fibrous union had occurred. As a result, the incus-strut union had not persisted and incus and strut were not in acoustic contact, the strut having been either dislocated partially from the incus or having fallen away, lying freely in the middle ear. This lack of incus-strut union was responsible in some cases for a departture of the medial aspect of the strut from its desired contact point with footplate or footplate fragment so that it became dislodged from the operative placement and ended up in contact with a fixed margin of oval window, usually in an area of otosclerotic bone where there was no possibility of transmission of acoustic energy to perilymph (fig. 29).

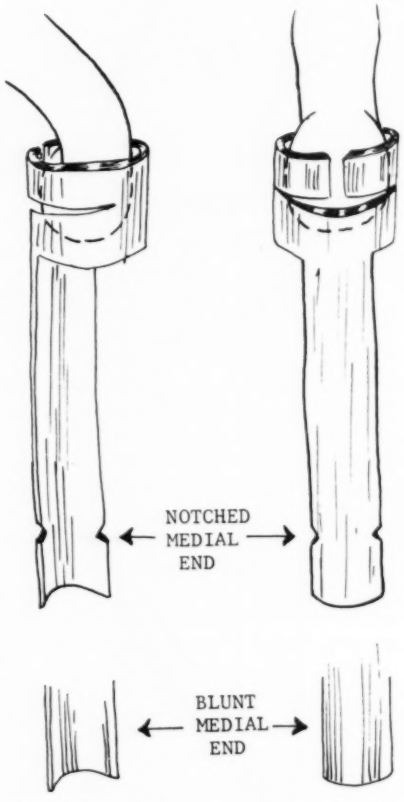

FIG. 27—Collar articulated prosthesis. Double ring at lateral end. External ring is slit to form collar which grasps long process as lenticular process fits into inner ring.

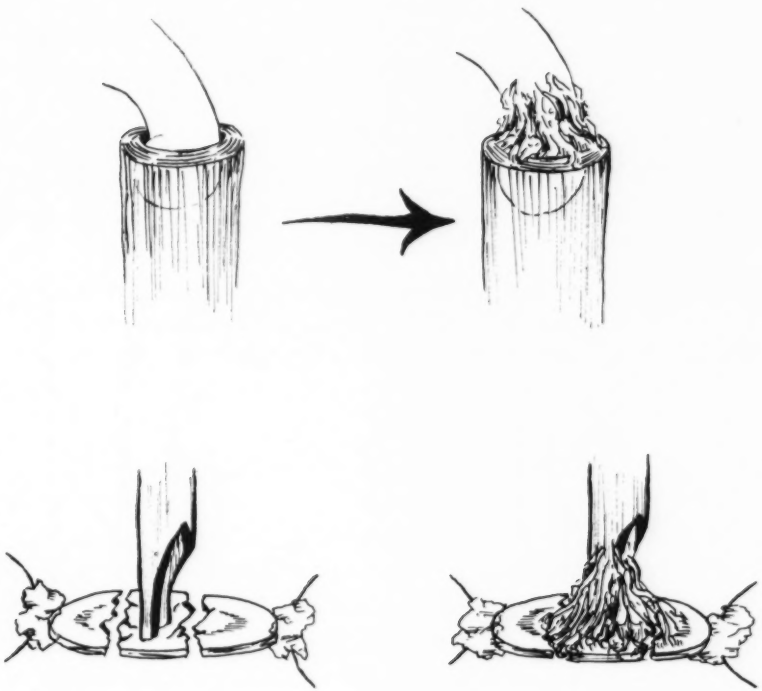

FIG. 28—Fibrous tissue fixed the incus-strut union and the strut-footplate union to recreate a functional push-pull dynamic.

The first step in attempting to correct this problem was the creation of four or five flange cuts at the lateral aspect to widen the lumen of a smaller gauge tube and thus provide frictional grasping contact with lenticular process (fig. 30). This was a definite improvement and allowed a greater degree of predictability to strut-incus union, based, however, upon the presence of a fairly definite lenticular process. However, this was not a total solution, since the lenticular process was so shallow in some cases that it did not permit the flanged tube to get a secure attachment. This inspired a welded tube segment forming a T-shaped prosthesis, with the horizontal portion of the T mounted on the incus. Several varieties of this version were created, some successful, others not.

The final stage in the evolution of the polyethylene strut to promote a permanent incus-strut union was the fashioning of the collar prosthesis. In this prosthesis the lateral aspect consists of two rings, the outer one slit open to form a locking collar. Polyethylene tubing, gauge 60, is used. The collar prosthesis utilizes the inherent elasticity or "memory" of polyethylene to create a partially encircling clasp (fig. 27).

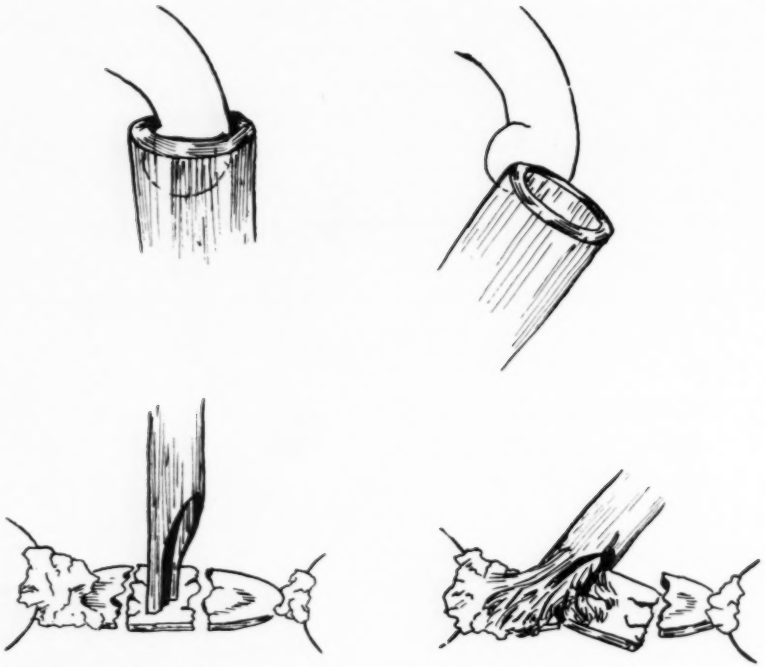

FIG. 29—Unsatisfactory incus-strut union with dislocation and wedging of medial end into fixed otosclerotic margin. No transmission is possible.

The collar is spread open with a specially designed strut introducer (figs. 31 and 32) so that the lenticular process is introduced into the inner ring segment (fig. 33). The introducer is then removed, allowing the collar clasp to snap shut (fig. 34) and thus obtain and maintain a firm but gentle hold on the terminal aspect of the long process of the incus.

The evolution of this collar prosthesis to its present state has created a virtually permanent crus-incus union which appears to be effective and safe. It is necessary, of course, for the prosthesis to be properly fashioned so that the collar has sufficient grasping force. If it is improperly constructed, it will not function as a clasp. It must grasp the incus for immediate push-pull dynamics to be an effective articulated prosthesis.

The elimination of the tubal aspect from the rest of the prosthesis has made it possible to direct the strut into a narrow area which previously did not admit a conventional, diagonally cut strut; particularly is this true in cases of massive otosclerotic infiltration of the promontory which may obliterate the caudal aspect of the fossa ovalis (oval window recess or niche) to a great degree. While it is possible, of course, to remove some of this otosclerotic bone with chisel or

FIG. 30—Flanging of medial strut end to allow grasping contact with lenticular process.

burr, such removal remains a questionable procedure, since no one can predict the osteogenetic sequelae of such radical dissection of otosclerotic bone.

Having solved the articulation problem with the collar, and the promontory friction problem with the narrow strut, only the medial end of the strut (the oval window "articulation") has remained an unsolved problem. While it is now possible to create a virtually completely articulated prosthesis at the incus end, an oval window articulation cannot be made with certainty at the time of the operative procedure but must be assumed to occur at some time later in the healing phase. That such unions may or may not occur is well known to all stapes surgeons. While there may be high and low degrees of predictability, there is no absolute guarantee that an oval window articulation will occur; thus, occasional failures with struts occur in all techniques advocated today.

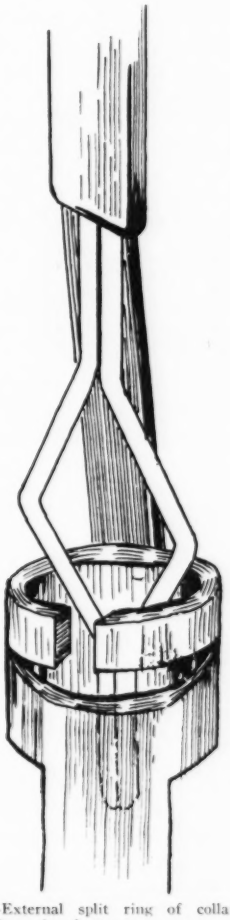

FIG. 31—External split ring of collar prosthesis with introducer in place.

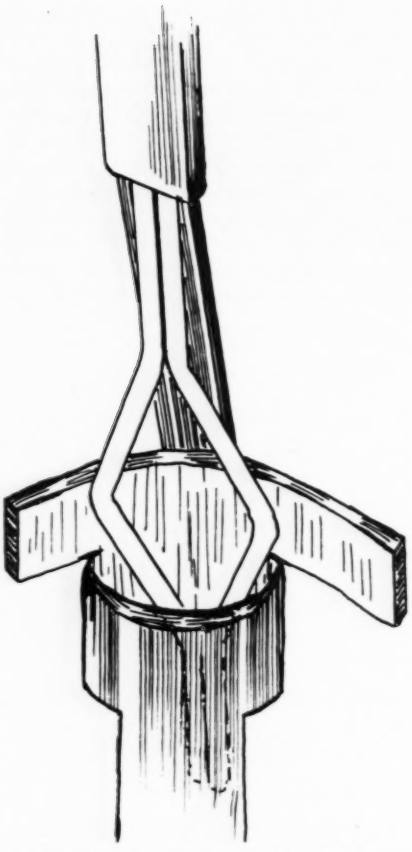

FIG. 32—Collar being spread by introducer.

At the present time we employ three types of medial ends to suit surgical requirements:

1. *The Blunt End*

The simplest type is a straight smooth-edged blunt strut 3.75 to 4.0 mm. long from inter-ring margin to medial end. This type is used when there is a completely mobile footplate following subtotal stapedectomy or when a large completely mobile footplate virgin island cracks loose with the slightest degree of pressure. In such instances, it is not necessary for the strut to enter the vestibular perilymph. Where a movable lid is in contact with the blunt end of a well articulated strut, this is quite adequate to ensure good transmission of acoustic energy. The length of the strut should be adequate for close approximation to the lid. In such instances, adequate physiologic reconstruction will be clearly

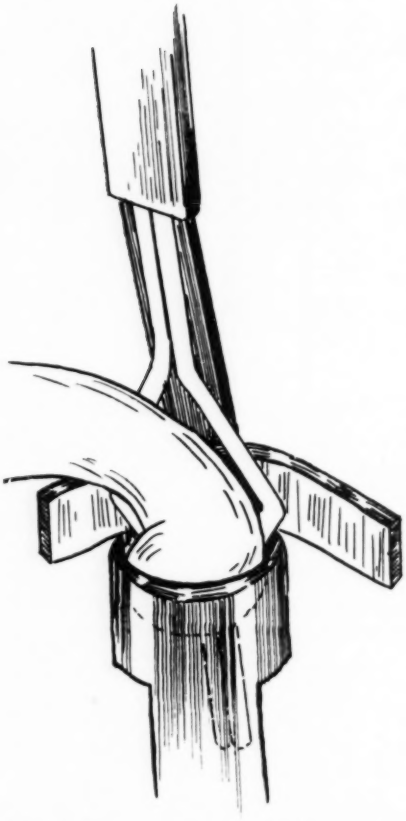

FIG. 33—Lenticular process fitting into inner ring while collar is open.

FIG. 34—Introducer being withdrawn. Collar snaps shut locking strut to incus for push-pull acoustic dynamics.

evident on surgical audiometry. It is obviously not necessary to resort to drastic procedures if a simple method will suffice (fig. 27).

2. *The Notched End*

The second version of the medial end is the "notched" version, in which two notches are placed 0.25 mm. from the medial tip of the strut. The total length of this strut is 4.00 to 4.25 mm. from inter-ring margin to medial tip (fig. 27). These notches are created to offer uneven surfaces to invite insinuation of the postoperative fibrous muco-endosteal membranes so that the strut will become incorporated in the healed mobile footplate segment. This is desirable in cases where a virgin island is present in an otherwise completely solid footplate. Fracture through such a virgin island will allow penetration of the notched medial end of the articulated strut into the vestibule. The fractured fragments of the virgin island are then covered by several pledgets of Hydeltrasol-saturated gelfoam. When healing takes place, the fibrous strands appearing between the footplate fragments become incorporated into the notches of the medial end of the strut so that the strut becomes a part of the now totally mobile virgin island.

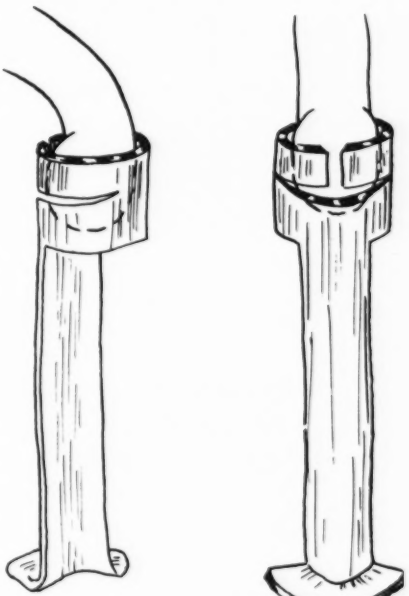

FIG. 35—Collar articulated prosthesis with blunt "foot" at medial end, for use in total stapedectomy either with fat or vein graft.

3. *The Foot End*

The third version of the medial end of the articulated prosthesis is the welded foot end which is used in total stapedectomy (fig. 35). The foot is formed by heat welding a 1.5 mm. wide segment of polyethylene tubing (90 degree wedge) to the blunt medial end of a Type I strut. Thus the total length is 4.00 to 4.25 mm. from inter-ring margin to medial end of foot.

The articulated prosthesis is first attached to the incus and is then rotated so that the foot of the prosthesis projects into the vestibule, resting on a membrane graft (figs. 36 and 37). In the case of a fat graft, it rests on the primary fat graft used to seal the oval window. It is then covered with two or three smaller pieces of fat. This fatty bed surrounds the foot and the medial end of the strut. The foot is incorporated in the eventual muco-endosteal membrane which replaces the fat graft. The prosthesis then functions as an integral portion of a newly created fibrous footplate substitute. Thus the danger of its slipping away from the tissue footplate is minimized because it already occupies an intravestibular position by virtue of the incus articulation, and placement of the fat graft medial and lateral to the foot encourages the creation of an enveloping muco-endosteal membrane. Thus one may expect a total push-pull articulation within a matter of several days as the new fibrous tissue or pseudo footplate forms.

The articulated prosthesis is equally effective in the vein graft technique. Either Type I (the blunt end) or Type III (the foot end) may be used. In the vein graft, however, there is the greater possibility of a non-union with the medial end than in the fat graft, which deliberately incorporates the strut into its substance.

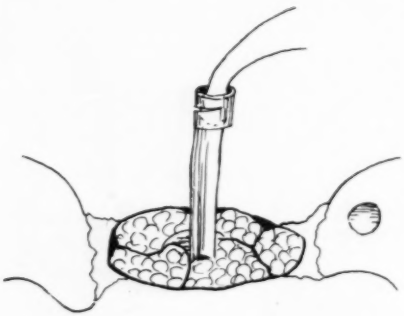

FIG. 36—Articulated prosthesis with foot covered by fat grafts.

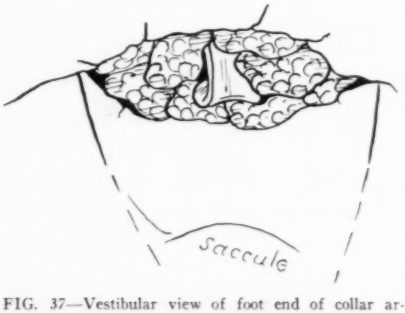

FIG. 37—Vestibular view of foot end of collar articulated prosthesis surrounded by fat grafts.

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STREPTOMYCIN SULFATE AND DIHYDROSTREPTOMYCIN TOXICITY

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THE primary toxic effect of streptomycin sulfate is manifested in man by dysfunction of the vestibular mechanism resulting in vertigo, ataxia and loss of postrotatory nystagmus.[1,5,9,11] In the case graphed in figure 1, toxic doses of streptomycin sulfate were deliberately administered to relieve vestibular symptoms of bilateral Ménière's disease. Note the loss of function of the lateral canal crista, as indicated by the complete loss of nystagmus on caloric stimulation with ice water.

The toxicity of dihydrostreptomycin at first eluded the clinical observer, since the toxic effects are delayed and may occur as late as several weeks to six months following the initial use of the drug.[6,7] This delayed toxic effect manifests itself as a sensorineural hearing loss which may involve any or all frequencies.

In August 1959, Shambaugh and his co-workers reported thirty-two cases of sensorineural deafness following the use of dihydrostreptomycin either in combination with other drugs or alone.[12] Twenty-five of these hearing losses were delayed one or more months following treatment. A representative audiogram of a patient who received dihydrostreptomycin and experienced the onset of a loss of hearing five months later is seen in figure 2.

A number of investigators have studied animals following induced streptomycin sulfate toxicity to observe the site of the organic lesions. Caussé[3] and Berg[2] described the degenerative changes in the vestibular sense organ and in the organ of Corti following toxic doses of streptomycin sulfate. Christensen and his co-workers[4] and Winston and his colleagues[13] concluded that organic changes were to be found in the vestibular neural pathways. Hawkins and Lurie[8] reported changes in the organ of Corti following dihydrostreptomycin intoxication in cats. No experimental evidence confirming the cause and effect relationship between dihydrostreptomycin and delayed hearing losses was reported.

THE PRESENT STUDY

A controlled study was designed to clarify further the site of the toxic effects of streptomycin and dihydrostreptomycin and to relate the histopathologic findings in the temporal bones and brain stem nuclei to alterations of vestibular and auditory function.

The experimental group consisted of thirteen healthy adult cats. In each cat, one ear had been surgically destroyed. Following recovery from this procedure,

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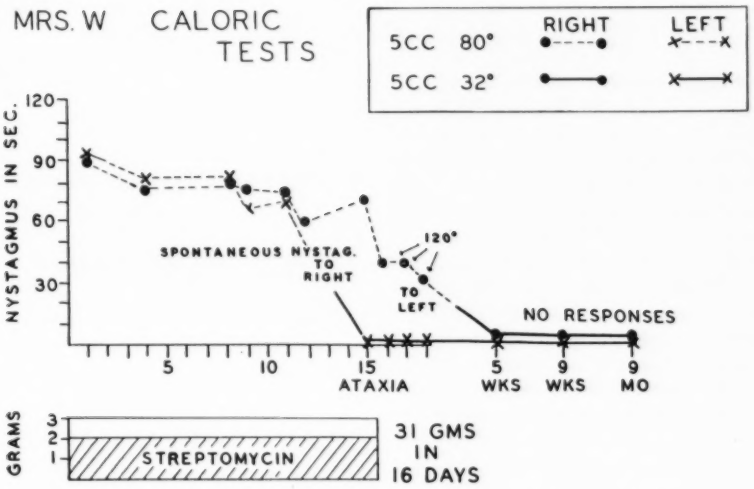

FIG. 1—Poststimulation nystagmus in seconds is plotted as a function of time in days for a patient treated with toxic doses of streptomycin sulfate.

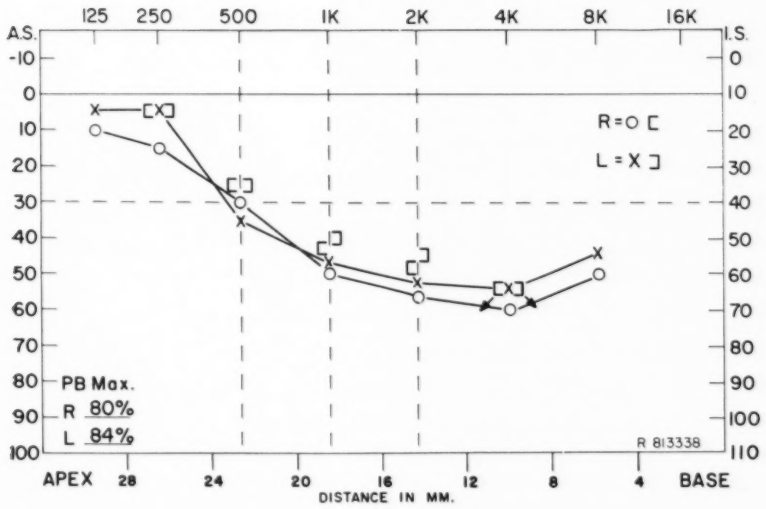

FIG. 2—Audiogram illustrating a delayed sensorineural hearing loss following treatment with 8 grams of dihydrostreptomycin.

base-line vestibular tests and audiograms were made. In the test of vestibular function the animal was placed on a rotating table with its head over the axis of rotation. The table was turned at the rate of one turn per second for ten seconds, then abruptly stopped. The duration of postrotational nystagmus was determined for turning both to the right and to the left. The results of this test, carried out in the horizontal plane of the head, indicate the functional state of the crista of the horizontal canal. Base-line cochlear function was evaluated by obtaining a series of reproducible audiograms for each animal before, during, and following treatment with the drug. These were done, utilizing avoidance conditioning techniques by which the animal was trained to avoid shock by moving forward in a rotary cage in response to auditory stimuli.[10] Because the object of the experiment was to study toxic effects, the dosage and duration of administration of the streptomycin and dihydrostreptomycin varied considerably in the different animals, with the functional tests serving as a monitoring method for determining the state of toxicity.

Seven animals were injected intramuscularly with streptomycin sulfate, amounts varying for the different animals from 25 to 200 mg. per kilogram of body weight each day in two equally divided doses. Six animals were injected with dihydrostreptomycin in amounts which varied from 100 to 300 mg. per kilogram of body weight each day in two equally divided doses. When the desired toxic effects for each drug had been obtained, the animals were kept for twelve to twenty months and then sacrificed by intravital perfusion. The temporal bones and brain stems were then prepared in the usual manner for pathologic study.

One representative animal from the streptomycin group and two representative animals from the dihydrostreptomycin group will be presented.

Results of Treatment with Streptomycin Sulfate

Cat 11 received 50 mg. of streptomycin sulfate per day for each kilogram of body weight. This dosage was continued for thirty-six days. No hearing loss was experienced by this animal, but there were a few outer hair cells missing in the upper basal turn, the absence of which did not influence the pure tone thresholds (fig. 3). The first order neurons of the organ of Corti were normal. The cat developed ataxia on the twenty-second day of treatment and the duration of the postrotatory nystagmus began to decrease on the twentieth day of treatment, going to zero on the twenty-fifth day (fig. 4, A). Twenty-two

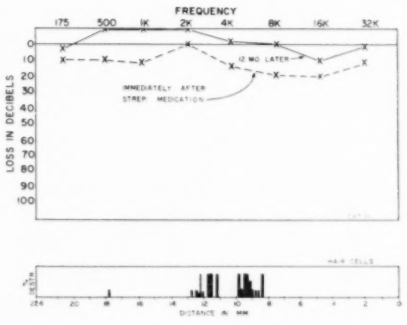

FIG. 3—Cat 11. Audiogram indicates no loss of hearing, either immediate or delayed, following streptomycin sulfate injections. Destruction of hair cells is indicated in black on the anatomical frequency scale below the audiogram.

days following the last injection there was partial return of the postrotatory nystagmus. The histopathologic findings were most marked in the cristae of the semicircular canals, with little pathologic change present in the maculas of the saccule and utricle (fig. 4, B). The

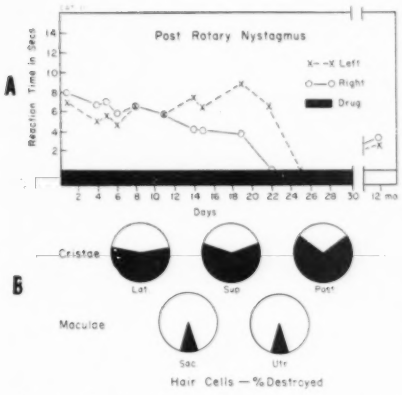

FIG. 4—Cat 11. (A) Duration of postrotatory nystagmus in seconds plotted as a function of time in days for a cat that received 50 mg. of streptomycin sulfate per day for each kilogram of body weight, as indicated. (B) Graphic representation of the histopathologic changes seen in the vestibular end organ, which are indicated in black.

histopathology of the cristae was one of loss of hair cells, flattening of the sensory epithelium and pyknosis and loss of cell membranes of the hair cells remaining (fig. 5).

Results of Treatment with Dihydrostreptomycin

Cat 23 received 200 mg. of dihydrostreptomycin per day for each kilogram of body weight, for sixty days. On the forty-fourth day of treatment the cat developed ataxia, and on the fortieth day the duration of postrotatory nystagmus began to decrease, going to zero on the fifty-fifth day, where it remained until the animal was sacrificed twenty months later (fig. 6, A). The histopathologic findings were most marked in the cristae of the semicircular canals and were identical to those observed in the cats receiving streptomycin sulfate (fig. 6, B). Audiograms done during the period of treatment and following the completion of treatment revealed a loss of hearing which occurred two months after administration of the drug and remained until the animal was sacrificed eighteen months later (fig. 7).

Cat 26 received 300 mg. of dihydrostreptomycin per day for each kilogram of body weight, for twenty-one days. The animal became ataxic on the six-

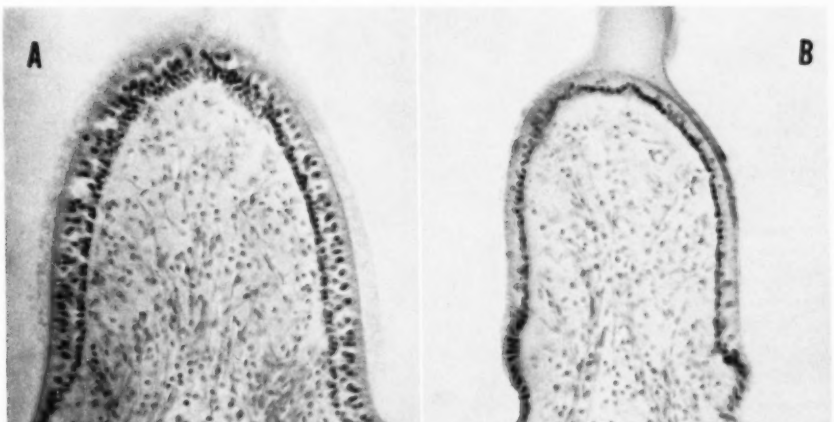

FIG. 5—(A) Photomicrograph of a normal lateral canal crista of the cat. (B) Crista of the lateral canal following treatment with toxic doses of streptomycin sulfate.

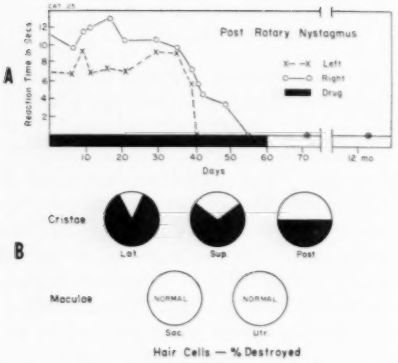

FIG. 6—Cat 23. (A) Duration of postrotatory nystagmus in seconds plotted as a function of time in days for a cat that received 200 mg. of dihydrostreptomycin per kilogram of body weight each day as indicated. (B) Graphic representation of histopathologic changes in the vestibular end organ, which are indicated in black.

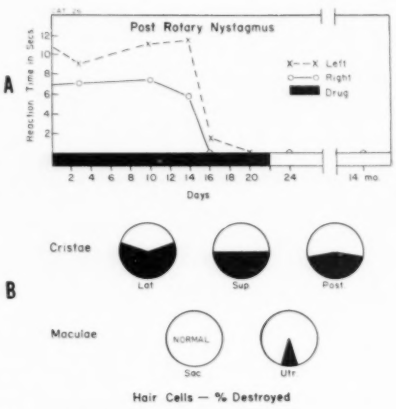

FIG. 8—Cat 26. (A) Duration of postrotatory nystagmus in seconds plotted as a function of time in days for a cat that received 300 mg. of dihydrostreptomycin per kilogram of body weight each day as indicated. (B) Graphic representation of histopathologic changes in the vestibular end organ, as indicated in black.

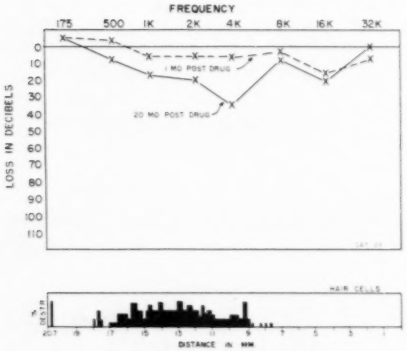

FIG. 7—Cat 23. Audiograms illustrating the presence of a permanent delayed loss of hearing following completion of dihydrostreptomycin injections. Hair cell pathology in black is plotted on the anatomical frequency scale below the audiogram.

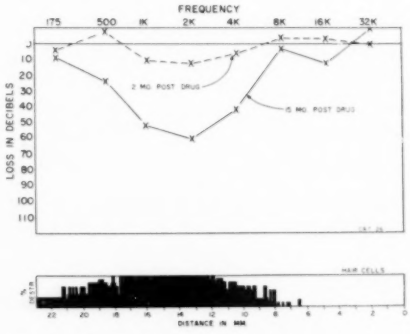

FIG. 9—Cat 26. Audiogram illustrating the presence of a permanent delayed loss of hearing following completion of dihydrostreptomycin injections. Destruction of hair cells is plotted in black on the anatomical frequency scale below the audiogram.

teenth day of treatment, and the duration of the postrotatory nystagmus went to zero on the twentieth day, where it remained until the animal was sacrificed fifteen months later (fig. 8, A). Again, the histopathologic findings in the vestibular end organ were most marked in the cristae of the semicircular canals and were identical to those observed in the two animals discussed above (fig. 8, B).

Audiograms obtained during the period of treatment and following the completion of treatment revealed a loss of hearing which occurred eight months af-

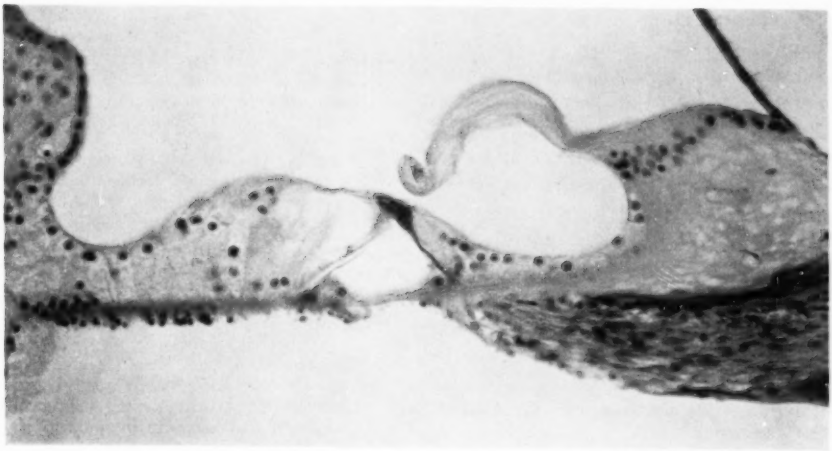

FIG. 10—Photomicrograph of the organ of Corti of an animal that received toxic doses of dihydrostreptomycin. Note complete absence of inner and outer hair cells.

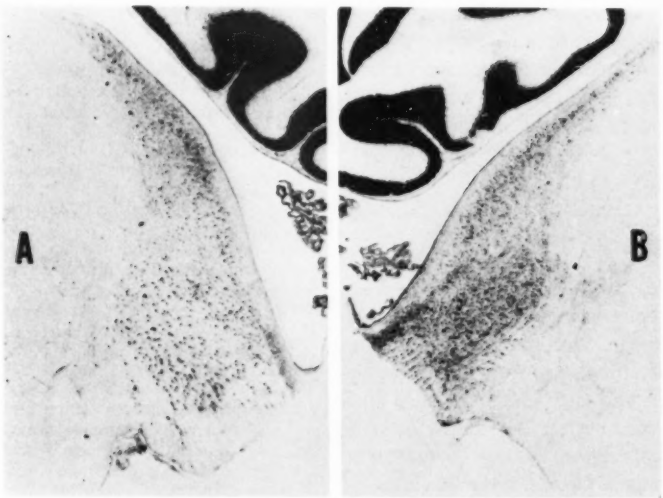

FIG. 11—(A) Photomicrograph of the dorsal and ventral cochlear nucleus of an experimental ear that experienced a delayed hearing loss following dihydrostreptomycin treatment. (B) Ventral and dorsal cochlear nuclei of labyrinthectomized ear of same animal. Note dense grouping of nuclei secondary to loss of cytoplasm from cell bodies.

ter administration of the drug, and remained until the animal was sacrificed seven months later (fig. 9). The histopathology present in the organ of Corti ranged from cloudy swelling and pyknosis to complete loss of inner and outer hair cells (fig. 10). The first order neurons of the hair cells were normal.

Effects in the Brain Stem

I am deeply indebted to Dr. J. Olszewski, Professor of Neuropathology at the University of Toronto, for his evaluation of the brain stem nuclei. Dr. Olszewski serially sectioned and studied twenty brain stems, which represented a coded mixture composed of thirteen experimental and seven control cats. The code was broken following the completion of the experiment. The dorsal and ventral cochlear nuclei and the vestibular nuclei of the experimental and control ears showed no evidence of pathologic change. The brain stem nuclei of the labyrinthectomized ears revealed changes compatible with surgical destruction (fig. 11).

CONCLUSION

The lesion in streptomycin and dihydrostreptomycin toxicity is limited to the inner ear. Streptomycin sulfate and dihydrostreptomycin both damage the vestibular end organ. They do so in a selective manner in the cat, where the cristae appear to be less resistant to toxic changes than do the maculas of the saccule and the utricle. Dihydrostreptomycin toxicity is associated with a delayed permanent loss of hearing which can occur several weeks to several months following treatment with the drug.

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DISCUSSION

GEORGE E. SHAMBAUGH, JR., M.D., Chicago: I wish to compliment Dr. McGee on this beautiful piece of work, well planned, expertly conducted and lucidly reported. Especially interesting to me is the experimental confirmation of the delayed cochlear loss that may follow dihydrostreptomycin medication by several months. This latent period between administration of the drug and the onset of hearing impairment is unique among all of the known ototoxic drugs. This was first pointed out by Glorig in 1951 and subsequently confirmed by other authors. Now that we have experimental confirmation of delayed hearing loss produced in the experimental animal by administration of dihydrostreptomycin the investigation of the mechanism of this unique and peculiar effect becomes a possibility. There has been uncertainty regarding site of injury. Dr. McGee's study points very clearly and definitely to the end organ in the labyrinth.

Clinically, dihydrostreptomycin deafness has been important because this drug has been included in many commercial combinations of antibiotics, the trade names of which failed to indicate the presence of an ototoxic drug. Bicillimycin, Cillimycin, Combiotic, Crysdimycin, Dicrysticin, Dihydrocillin, Distrycillin, Durycin, SRD and Strocillin all contained between one-quarter and one gram of dihydrostreptomycin with 400,000 to 900,000 units of penicillin per dose. In some cases the antibiotic was given not as a life-saving measure for serious diseases but for a mild self-limiting infection, such as the common cold, or prophylactically to uninfected surgical patients. The prescribing physician usually had no idea of the risks to the hearing, and since the deafness began weeks or months later, he was not aware of the irreversible hearing loss when it developed: We must remember the marked differences in individual susceptibility. In some cases the deafness comes on immediately, but many cases are noteworthy because of a latent period of weeks or months during which there is normal hearing. This is followed by a fairly rapid onset of a neurosensory or nerve deafness which soon reaches a plateau, where it levels off. This hearing loss is irreversible and is characterized by a disproportionate impairment of discrimination, so that a hearing aid is of limited or no help.

One year ago under the sponsorship of the Committee on the Conservation of Hearing, the article mentioned by Dr. McGee was published in the *Journal of the American Medical Association*, calling attention to this risk. We can take at least partial credit for the subsequent withdrawal by drug companies of dihydrostreptomycin from various commercial combinations of antibiotics.

Since streptomycin is equally effective against gram-negative bacteria, and in effective therapeutic doses is safe as far as hearing is concerned, there is no real reason for continuing the manufacture and sale of dihydrostreptomycin. We may hope that deafness from this source will soon become a thing of the past. However, we must keep in mind that new antibiotics may prove to have an unsuspected ototoxicity which will be discovered only by alertness on our part.

A SURGICAL OTOSCOPE

BEN H. SENTURIA, M.D.
ST. LOUIS, MISSOURI

CURRENT studies[10] reveal that strict postoperative asepsis is necessary in the care of the external auditory canal and middle ear if we are to be successful in stapes and middle ear reconstructive surgery. Implantation of exogenous organisms during the postoperative period can vitiate all the good results obtained through meticulous microsurgery.

Furthermore, it has become increasingly evident that magnification and good light are essential to the performance of myringotomy and the aspiration of middle ear effusions. If we are to remove, aseptically, thick mucopurulent secretions from the middle ear with a minimum of trauma to the tympanic structures, it is mandatory that proper instrumentation be available.

It is neither novel nor unusual for the medical practitioner to seek better means of visualizing the external and middle ears. One finds mention of the use, in India, of a speculum for studying the ear as early as the first century of the Christian era.[2]

The problem of adequate illumination was a major obstacle. In 1363 Guy de Chauliac, with information obtained from the Arabians, used sunlight to aid in examining the ear with a speculum.[5] Later, in 1587, Guilio Cesare Aranzi described the use of artificial light for visualizing the nasal orifices.[1] Archibald Cleland,[6] in England (1740-1741), stated:

> In order to discover, with more exactness, whether the disorder lies in the outward ear, I make use of a convex glass, three inches in diameter, fixed in a handle into which is lodged some wax candle, which comes out at a hole near the glass, and reaches to the centre; which, when lighted, will dart the collected rays of light into the bottom of the ear. . . .

Bozzini later (1807) wrote a classical treatise with beautifully executed illustrations of the apparatus and adapters for illuminating, with artificial light, the various "inner cavities and interstices of the living animal body."[4]

The earliest electric otoscope, described by Hedinger in 1879, utilized platinum wires attached to a finely polished hemispheric mirror and derived its current from a battery.[7] In 1884, Rosewell Park presented a similar instrument.[9] Many more contributions may be found in the literature up to the time that Lüscher[8] and Blackmore and Hallpike[3] described their intricate otoscopes for magnifying and illuminating the tympanic membrane and middle ear. Needless to say, the recent introduction of the Zeiss binocular otomicroscope, which provides coaxial light and interchangeable magnifying lenses and allows adequate operating space, revolutionized otologic surgery.

From the Departments of Otolaryngology, Washington University School of Medicine and The Jewish Hospital, St. Louis, Missouri.

This investigation was partially supported by funds provided from the St. Louis Otological Foundation.

Presented as a *New Instrument* at the Sixty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1960, Chicago.

Appreciation is expressed to the Storz Instrument Company, St. Louis, Missouri, for their cooperation in the development of this instrument, and to the Smithsonian Institute, Washington, D. C., for supplying source material.

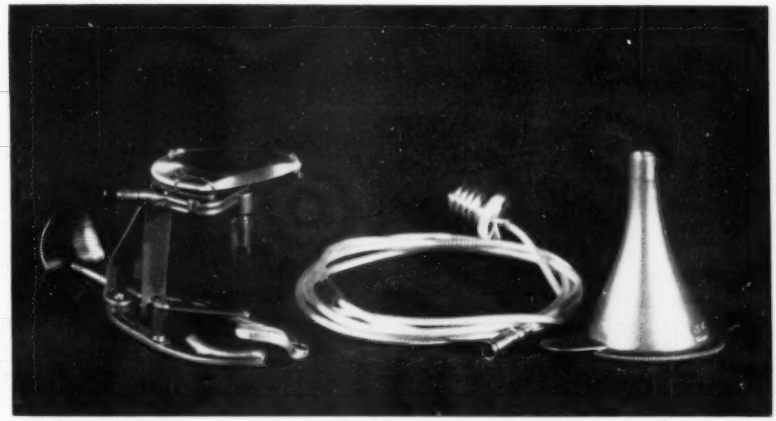

FIG. 2—Side view showing surgical otoscope, battery cord and speculae.

Despite the great progress made in the development of the otoscope, the practicing otologist has not availed himself of the benefits of these advances; the Zeiss otomicroscope is too expensive and cumbersome for routine office work, and the Hallpike-Blackmore instrument does not readily lend itself to the care and treatment of sterile postoperative cavities. To my knowledge, none of the available smaller otoscopes can be satisfactorily sterilized.

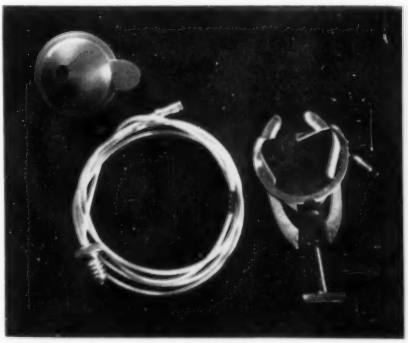

FIG. 1—View from above showing surgical otoscope, battery cord and speculae.

This new instrument (figs. 1 and 2) introduces the following practical advantages to the otologist:

1. The instrument is simple, light, compact and rugged in structure. The entire instrument, including the lens, light bulb and battery cord, may be autoclaved.

2. The built-in illumination eliminates the need for expensive nonexplosive lamps as a source of reflected light and removes the frequently used unsterile examining otoscope from the sterile operating field.

3. A sterile speculum of the desired size may be inserted easily and quickly without the use of rubber gloves.

4. The single magnifying lens may be removed for purposes of sterilization and permits each otologist to incorporate any desired optical correction.

5. The magnification included (2.5×) is sufficient for all routine office diagnostic procedures and gives the re-

quired detail for myringotomy and aspiration.

6. The instrument permits the use of a variety of light sources presently in use (e.g., the otoscope battery handle) and many varieties of speculae, myringotomy knives, curettes and applicators which are already available in the otologist's office.

7. Myringotomy, biopsy, aspiration of secretions and removal of packing may be accomplished with complete sterility in the office or operating room.

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A MIDDLE EAR INTUBATOR

ALBERT H. WILLIAMS, M.D.
FITCHBURG, MASSACHUSETTS

IT is not my intention to extol the virtues or debate the use of myringotomy versus intubation in the management of chronic serous otitis media, but for those who agree that fenestration of the tympanic membrane has a place in our armamentarium for this condition, I wish to proffer this device.

This new instrument is a modification of the angulated Krause type of aural polyps snare with which we are all familiar. The wire carrier has been removed and a sliding cutting tube substituted (fig. 1, left). The tube resembles the hollow shaft of a spinal needle which has been closely beveled and sharpened.

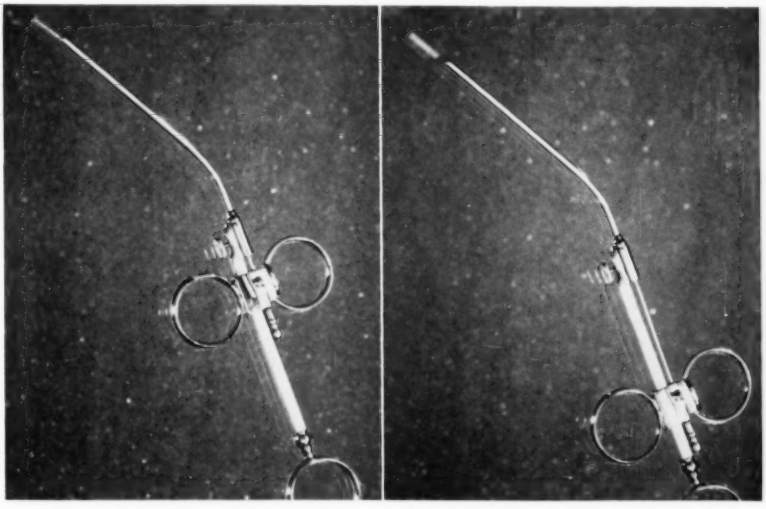

FIG. 1—(Left) Plastic tube attached. (Right) Plastic tube disengaged.

A short length of either polyethylene or polyvinyl chloride tubing (approximately eight or ten millimeters) is now slipped over the metal cutting tube so that it covers about one or two millimeters (fig. 1, right). The plastic tube

I wish to express my appreciation to Mr. Eric Storz of St. Louis, who collaborated with me in the development of this instrument.

Presented as a *New Instrument* at the Sixty-Fifth Annual Session of the American Academy of Ophthalmology and Otolaryngology, Oct. 9-14, 1960, Chicago.

has previously been prepared for use by flaring the connecting end by holding it over an alcohol lamp while rolling it between the fingers. The other end is cut on the bias so that it will not become occluded if pushed against the promontory.

The instrument is introduced into the external auditory canal under direct vision, and with a slight thrusting motion the drum is pierced. The thrust carries the plastic tube through the fenestration and, upon retraction of the metal tube by a finger pull on the ring, the instrument is disengaged, leaving the plastic tube in situ. Half of the tube remains in the tympanum; the distal half lies in the external auditory canal. Anesthesia is usually topical, as the procedure is well tolerated and generally bloodless.

I have reported on the current model. It is not inconceivable that a double-barreled modification may appear soon for those who prefer to introduce two tubes simultaneously.

TRANSACTIONS

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William L. Benedict, M.D., LL.D., Executive Secretary-Treasurer
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15 Second Street S.W., Rochester, Minnesota

COMMITTEE ON CONSERVATION OF HEARING SUBCOMMITTEE FOR LISTING OF AUDIOMETERS: LISTING PROCEDURE AND AGREEMENTS

1. *Advertising*

Manufacturers of audiometers may advertise the fact that their instruments are "listed" as passing the minimum requirement of the American Standards Association (ASA) for a pure tone audiometer, a limited frequency audiometer or a speech audiometer as the case may be. To date the usual wording has been a variation of, "Listed by the American Academy of Ophthalmology and Otolaryngology as conforming to the minimum standards of the American Standards Association for an..... audiometer"; or simply, "Listed by the American Academy of Ophthalmology and Otolaryngology." The terms "approved" and "certified" are not to be used and no official seal or other mark of approval will be authorized by the Academy.

2. *Exhibits*

It has been agreed that although a manufacturer could exhibit a new audiometer at an annual meeting of the Academy, he would not be permitted to do so for more than one year unless the instrument had passed the requirements for listing.

3. *Requirements for Listing*

The manufacturer must have submitted a production line instrument picked out at random by an agent of the Subcommittee, for testing. In cases where a superficial flaw is discovered in the production instrument selected by chance, such minor matters may be ironed out informally between the referee laboratory and the manufacturer before formal listing is announced or denied.

4. *Choice of Model*

Manufacturers are advised to submit for listing the most complicated model of a given basic design so that tacit listing of less complicated instruments can be obtained without the time and expense that would be necessary to test each lesser model. It is an almost invariable rule that if a complicated variant of a given basic design will pass the test simpler variants will also pass.

5. *Costs of Testing*

Manufacturers must agree to pay all shipping charges and to have their instruments tested on a "cost plus basis." They must pay a $400.00 minimum testing fee in advance. If special features of the instrument are to be tested, such as masking devices and the like, an additional $50.00 to test each such feature will be added. More elaborate devices attached to a pure tone audiometer to convert it into a speech audiometer, for example, will undoubtedly cost more. If testing of auxiliary equipment is desired, it must be so specified.

6. *Method of Procurement*

The manufacturer must name several dealers from whom a production line instrument for testing can be obtained at random or specify some other way that it can be obtained by an agent of the Subcommittee. Dealers should be apprised of the fact that when an instrument is selected they will receive an official letter from the American Academy of Ophthalmology and Otolaryngology in lieu of payment. They should then arrange the accounting and inventory problem with the manufacturer involved and arrange for shipping to a designated testing laboratory. After testing the manufacturer will be asked to send shipping instructions to the referee laboratory for return of the test instrument.

7. *Manufacturers Acceptance*

Acceptance means that the audiometer manufacturer has demonstrated the ability to produce one instrument that performs according to ASA specifications. It does not imply that any individual instrument will perform within the tolerances specified. It is obviously beyond the scope of the Listing Committee or the referee laboratories to provide production control for audiometer manufacturers and their customers. However, listing does indicate that the product can come up to ASA specifications and it is up to the customer to carry on negotiations with the manufacturer if a particular instrument which he receives is faulty.

January 31, 1961

ACADEMY PLANNED TOUR

At the request of a number of Academy members a suggested itinerary to the XIX International Congress of Ophthalmology and around the world (December 2 to 7, 1962) has been especially prepared for the American Academy of Ophthalmology and Otolaryngology by the Travel Department of the American Automobile Association in association with the Trans World Airlines.

The tour is planned on regular scheduled flights, jet economy, beginning November 18, 1962, at New York and ending December 21, 1962, at Honolulu. The services of a tour conductor will be included from arrival in Paris until departure from Tokyo. All rail and steamship transportation will be first class and reserved seats on trains are provided wherever available. Hotel accommodations are specified in the itinerary, including twin bedded rooms with bath.

This tour has been especially planned for the Academy by local representatives of the A.A.A. who have made many tours to the far East and have personal knowledge of the travel facilities available, and we can be assured of the very best.

The tour is officially terminated at Honolulu anticipating that several may choose to remain in the islands over the holidays.

The following countries will be visited with the assistance of the tour conductor, who will handle all matters of transpor-

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The Courses begin on September 1 each year and run continuously for ten months. Enrollment should be completed before August 15.

Application blanks and information may be secured from the Executive Secretary-Treasurer of the Academy: Dr. William L. Benedict, 15 Second Street S.W., Rochester, Minnesota.

tation and hotels: France, Italy, Egypt, India, Thailand, Hong Kong, Japan and Hawaii.

Complete arrangements can be made for the entire trip. For further information write A.A.A. World Wide Travel Department, 316 First Street, S. W., Rochester, Minnesota.

LETTER TO THE EDITOR

January 19, 1961

Dear Dr. Benedict:

I recently returned as the first staff ophthalmologist assigned to Project Hope in Indonesia. At that time I found a great lack of visual aids and teaching materials in the field of ophthalmology.

I am wondering if you would have any suggestions as to how we could best accomplish the donation of teaching aids such as prepared by the American Academy of Ophthalmology and Otolaryngology to these institutions. I know that they would be extremely worth while to the Indonesians and would do much to cement our friendship with the Indonesian doctors.

Also, back issues of the TRANSACTIONS or abstracts and teaching manuals would be most appreciated. The film "Embryology of the Eye" together with slides too would be, I feel, most worth while as a teaching endeavor.

Any thoughts that you might have as to how this might best be accomplished with little or no cost would be most appreciated. Looking forward to hearing from you.

Sincerely,

[Signed] DAVIS G. DURHAM, M.D.
The Professional Building
Augustine Cut-Off
Wilmington 3, Delaware

BOOKS FOR ASIAN STUDENTS

21 Drumm Street
San Francisco, California
March 23, 1961

Dear Dr. Benedict:

A few months ago we received a request from Dr. Wong Poh Lam of the Eye Department, General Hospital, Penang, Malaya for books and journals on ophthalmology. We had in our stock only Town's OPHTHALMOLOGY, which we sent to this hospital's library.

As you may know, The Asia Foundation's Books for Asian Students Program has shipped nearly two million books and over 300,000 journals to over 5,000 institutions in Asia during the past six years. We have been able, through the cooperation of various American groups and individuals, to supply books to meet specific reference needs of libraries in this area. Copies of the Foundation and book program brochures describing the work of both The Asia Foundation as a whole and its book program are enclosed.

We are writing to you specifically in the hope that some of your membership would be willing to donate recent and useful books and runs of journals for the Eye Department of the Penang General Hospital. We would, of course, need only one copy of each book and each journal run. We would appreciate having as long runs as possible of each journal, in any shipments made.

We would appreciate your consideration of informing your membership of this need, perhaps through an Academy house organ. As you will see on the last page of the book program brochure, The Asia Foundation will pay for transportation costs of materials sent in one of the two ways specified from the donor to San Francisco and thence to Penang.

Thank you for this consideration.

Sincerely yours,

(Signed) CARLTON LOWENBERG
Chief, Book Program

In Memoriam

Truman Y. Burton.....Colorado Springs, Colorado
December 29, 1960

Phillip H. Decker.....Williamsport, Pennsylvania
December 8, 1960

Frank C. Furlong.....Schenectady, New York
February 12, 1961

Rex E. Greer.....Amarillo, Texas
November 7, 1960

Ernest C. Hillyer.....Los Angeles, California
December 28, 1960

William P. Hofmann.....Davenport, Iowa
October 23, 1960

Roy Earl Mason.....St. Louis, Missouri
November 20, 1960

Gervais W. McAuliffe.....Bridgehampton, New York
October 17, 1960

Carroll R. Mullen.....Philadelphia, Pennsylvania
February 1, 1961

Elmer W. O'Brien.....Webster, New York
September 25, 1960

John J. Pagliughi.....Union City, New Jersey
February 15, 1961

Carlos E. Pitkin.....Cleveland, Ohio
February 24, 1961

John Frederick Sarno.....Johnstown, New York
November 6, 1960

Walter Knapp Slack.....Saginaw, Michigan
November 13, 1960

Dewey St. John.....St. Louis, Missouri
November 4, 1960

John Malcolm Tindal.....Chicago, Illinois
October 20, 1960

Frederick Turnbull.....Los Angeles, California
January 13, 1961

David Ray Womack.....Austin, Texas
August 26, 1960

LISTING OF AUDIOMETERS

The following audiometers are recommended for listing by a subcommittee of the Committee on Conservation of Hearing, American Academy of Ophthalmology and Otolaryngology:

PURE TONE AUDIOMETERS

Beltone Model 10-C Model 15-C	Beltone Hearing Aid Company Audiometer Division 2900 West 36th Street Chicago 32, Illinois
Maico Model MA-5 Model H1-B for pure tones only	Maico Electronics Inc. Maico Building 21 North Third Street Minneapolis 1, Minnesota
Sonotone Model 91	Sonotone Corporation Elmsford, New York
Zenith ZA 200	Zenith Radio Corporation 6501 West Grand Avenue Chicago 35, Illinois

SPEECH AUDIOMETERS

Grayson-Stadler Model 162	Grayson-Stadler Co. West Concord, Massachusetts

SCREENING AUDIOMETERS

Ambco Otometer 600	Ambco Inc. 1222 W. Washington Blvd. Los Angeles 7, California

Each model number in the list as a rule indicates the most advanced model of that series. If less advanced instruments are for sale under similar model numbers, it is assumed that they will function as well or better than the more complicated model in a series.

Acceptance for listing means that the audiometer manufacturer has demonstrated the ability to produce one instrument that performs according to A.S.A. specification. It does not imply that any individual instrument will perform within the tolerances. It is obviously beyond the scope of the Listing Committee or the Referee Laboratories to provide production control for audiometer manufacturers and their customers; however, listing does indicate that the product of the manufacturer can come up to 1951 A.S.A. specifications, and it is up to the customer to carry on negotiation with the manufacturer if a particular instrument which he receives is faulty.

Abatron D-100 and Ambco Oto-chek 800 were removed from the above list at the request of the respective manufacturers.

NEWS AND ANNOUNCEMENTS

U. S. DEPARTMENT OF HEALTH, EDUCATION AND WELFARE PUBLIC HEALTH SERVICE

A new program to support research training in neurosurgery through postgraduate training grants from the National Institute of Neurological Diseases and Blindness was announced on January 20, 1961, by Dr. Leroy E. Burney, Surgeon General of the U. S. Public Health Service.

The neurosurgery grants will be made to fill a need for qualified teachers and investigators in neurosurgery who will devote themselves to academic careers. Application forms for these grants can be obtained from the Chief of NINDB Extramural Programs, National Institutes of Health, Bethesda 14, Maryland.

The program marks the opening of another important phase in NINDB's development of postgraduate research training in medical specialties. It complements similar programs underway at a number of institutions throughout the nation. Current support includes grants for research training programs in neurology, ophthalmology, otolaryngology, neuroanatomy, neurochemistry, neuropathology, neurophysiology, neuroradiology, medical audiology, sensory physiology, and pediatric neurology.

The National Institute of Neurological Diseases and Blindness, which is making grants for these research training programs, is one of the seven Institutes that make up the National Institutes of Health, the research arm of the U. S. Public Health Service.

IRISH OPHTHALMOLOGICAL SOCIETY

The Irish Ophthalmological Society will hold its Annual Meeting in Dublin on April 27 to 29, 1961. The Montgomery Lecture will be given by Professor Jules Francois of Ghent, Belgium, in the Royal College of Surgeons, Dublin, on Thursday, April 27.

HENRY HOLLAND HOSPITALS ALUMNI ASSOCIATION

Roland I. Pritikin, M.D., Rockford, Illinois, President of the Henry Holland Hospitals Alumni Association and Fund, addressed the Kobe (Japan) Society of Ophthalmology, the Hong Kong Ophthalmological Society, the Philippine Ophthalmological Society, the Department of Ophthalmology, University of Philippines, and other ophthalmological groups in the Far East during January 1961.

CASSELBERRY AWARD

A sufficient fund having accrued from the Casselberry Fund for encouraging advancement in the art and science of Laryngology and Rhinology, this sum is now available, in part or as a whole, for a prize award. Theses must be in the hands of the Secretary of the American Laryngological Association prior to December 1 of any given year.

The Award is a prize of money with accompanying certificate signed by the officers of the American Laryngological Association. The sum of money will be agreed upon by the Council of the Association after the manuscript has been evaluated by the Award Committee. It may be awarded in whole or in part among several contestants.

Eligible contestants may be (1) hospital interns, residents, or graduate students in Rhinology and Laryngology; (2) an individual with a degree as Doctor of Medicine who is actively practicing or teaching Rhinology and Laryngology in the Americas; (3) any scientific worker in the field of Rhinology and Laryngology.

Manuscripts shall be presented to the Secretary of the Association under a nom de plume which shall in **no way** indicate the author's identity. There shall also be a sealed envelope bearing the nom de plume and containing a card showing the name and address of the contestant, which the Secretary shall keep in his possession.

Manuscripts must be limited to 5000 words and must be typewritten in double spacing on one side of the sheet. The thesis shall not have been published elsewhere before submission.

The successful thesis shall become the property of the American Laryngological Association, but this provision shall in no way interfere with publication of the thesis in the journal of the author's choice. Unsuccessful contributions will be returned promptly to their authors.

The Award, which will be made at the Annual Meetings of the American Laryngological Association, shall be based on:

1. Originality of material
2. Scientific and clinical value
3. Suitability for this Award
4. Method of presentation as to style, illustrations and references

The maximum amount of the Award shall not exceed $200.00.

For further information write to Lyman G. Richards, M.D., Secretary, American Laryngological Association, 12 Clovelly Road, Wellesley Hills 81, Mass.

CHICAGO OPHTHALMOLOGICAL SOCIETY

The Chicago Ophthalmological Society will hold its annual clinical conference on May 19 and 20, 1961, at the Drake Hotel in Chicago. The following program has been arranged:

Address of Welcome, Warren F. Smith, M.D., Oak Park, Ill.

Symposium on Therapy of Primary Glaucoma
Medical Therapy of Primary Glaucoma, Bernard Becker, M.D., St. Louis

Surgical Therapy of Open Angle Glaucoma, Harold G. Scheie, M.D., Philadelphia

Surgical Therapy of Narrow Angle Glaucoma, R. N. Shaffer, M.D., San Francisco

Treatment of Retinal Detachment by the Equatorial Ring, Count H. Arruga, Barcelona, Spain

Some of the Factors Which Determine the Success of a Perforating Corneal Graft, Mr. A. G. Leigh, London, England

A Method of Corneal Grafting, Mr. A. G. Leigh

Symposium on Secondary Glaucoma
Role of Iritis, Bernard Becker, M.D.

Clinical Aspects and Treatments of Acute and Sub-acute Phacogenic Glaucoma, Mr. A. G. Leigh

Role of Postoperative Glaucoma, R. N. Shaffer, M.D.

The Problem of the Mobility of Post-enucleation and Post-evisceration Prostheses, Count H. Arruga

Cataract Extraction following Filtering Operations, Harold G. Scheie, M.D.

Roundtable discussions are planned for luncheon meetings on Friday and Saturday, and there will be periods for questions and answers on each day. On Friday, May 19, at 5:30 p.m., Dr. Harold G. Scheie will present the Seventeenth Annual Sanford R. Gifford Memorial Lecture. The title of his presentation will be *A New Forme Fruste of Hurler's Disease (Gargoylism).*

Registration fee for the entire course including roundtable luncheons and dinner following Gifford Memorial Lecture is $45.00, and is payable to Registrar, Mrs. Mary E. Ryan, 1150 N. Lorel Avenue, Chicago 51, Ill.

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

The Kansas City Society of Ophthalmology and Otolaryngology will hold its annual out-of-town meeting at the Hotel La Fonda, Santa Fe, New Mexico, March 23 to 25, 1961. The following program has been arranged:

OPHTHALMOLOGY

Office Management of Glaucoma, Albert N. Lemoine, Jr., M.D., Kansas City, Kansas

Glaucoma and Cataracts—Problems in the Etiology and Treatment of the Associated Conditions, Earl G. Padfield, M.D., Kansas City, Kansas

Management of Cataract Postoperative Complications, Albert N. Lemoine, Jr., M.D.

Ocular Injuries, Harold G. Scheie, M.D., Philadelphia

Surgical Procedures for Retinal Separations, Albert N. Lemoine, Jr., M.D.

Cataract Extraction, Wound Closure, and Experiences with Alpha Chymotrypsin, Harold G. Scheie, M.D.

Discussion of Fundus Photographs, Truman B. Schertz, M.D., Kansas City, Missouri

Discussion of External Disease Photographs, Max S. Lake, M.D., Salina, Kansas

OTOLARYNGOLOGY

Presbycusis in Old Age, C. P. Goetzinger, M.D., Kansas City, Kansas

Hearing Aids, C. P. Goetzinger, M.D.

Serous Otitis Media, Gunnar O. Proud, M.D. Kansas City, Kansas

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POSTGRADUATE COURSES

Baylor University College of Medicine

Baylor University College of Medicine, Texas Medical Center, Houston, Texas, in affiliation with Retina Foundation and Retina Service, Massachusetts Eye and Ear Infirmary, Boston, Massachusetts, announces a postgraduate course in ophthalmology, "Recent Developments in Ophthalmology." This meeting will be held June 5 to 7, 1961, at Baylor University College of Medicine, Texas Medical Center, Houston 25. Lectures will correlate recent developments in research with clinical management or diseases of the cornea, vitreous and retina. The doctors attending this meeting are invited to bring along patients that present a diagnostic problem that these may be presented for the attending group's opinion.

Guest speakers from the Retina Foundation will be Charles L. Schepens, M.D.; Endre A. Balazs, M.D.; Claes-Hendrik Dohlman, M.D., Ph.D., and Marie A. Jakus, M.D. Participating Baylor University College of Medicine Faculty will be Louis J. Girard, M.D.; Louis Daily, M.D., and Alice R. McPherson, M.D.

For application forms please write to Department of Ophthalmology, Baylor University College of Medicine, Texas Medical Center, Houston 25.

University of Michigan Medical School

The Ophthalmology Postgraduate Conference Program of the University of Michigan will be held April 24 to 26, 1961, in the Amphitheatre, third floor, Horace H. Rackham School of Graduate Studies. This conference will be conducted by Professor F. Bruce Fralick and staff and has been arranged for physicians who are especially interested in ophthalmology. It includes a review of fundamental principles by physicians of national prominence, as well as an opportunity to observe the application of modern developments in this specialty.

The fee for the conference is $50, payable upon application. Checks should be made to The University of Michigan. If the applicant is unable to attend, the fee will be returned.

The following program has been arranged:

Surgical Treatment of Certain Lid Disorders, S. D. McPherson, Jr., M.D., Durham, N. C.

Syndromes and Lesions of the Optic Nerve and Chiasm, Robert W. Hollenhorst, M.D., Rochester, Minn.

Management of Trauma of the Eyes in Children, Warren A. Wilson, M.D., Los Angeles

Medical and Surgical Management of the Patient with Chronic Open Angle Glaucoma, S. D. McPherson, Jr., M.D.

Interesting and Unusual Lesions of the Ocular Fundi, Robert W. Hollenhorst, M.D.

Congenital Cataracts in Children, Warren A. Wilson, M.D.

Ocular Pathology Before, In, and Following Retinal Detachment, J. Reimer Wolter, M.D., Ann Arbor, Mich.

Some Useful Techniques in Cataract Surgery, S. D. McPherson, Jr., M.D.

Complications of Ptosis Surgery, Warren A. Wilson, M.D.

Nystagmus, Robert W. Hollenhorst, M.D.

Complications of Ptosis Surgery, Warren A. Wilson, M.D.

Tumors of the Eye and Adnexa in Children; Diagnosis and Treatment, Charles E. Iliff, M.D., Baltimore

Diagnostic Ophthalmoscopic Changes, Harold F. Falls, M.D., Ann Arbor, Mich.

Surgical Technique, Charles E. Iliff, M.D.

Contact Lens, Charles E. Iliff, M.D.

On Tuesday evening, April 25, at a joint meeting of Detroit Ophthalmological Club, Detroit Ophthalmological Society, and Michigan Triological Society, Dr. F. Bruce Fralick will present the *Walter R. Parker Lectureship; Epidemiology of Suspected Etiology of Uveitis.*

Further information and application blanks may be obtained from John M. Sheldon, M.D., Director, Department of Postgraduate Medicine, The University of Michigan Medical School, Room 1610, University Hospital, Ann Arbor.

Far East Graduate Course In Ophthalmology

The first Far East Graduate Course in Ophthalmology under the auspices of the Ophthalmological Society of the Philippines and the Philippine Ophthalmological and Otolaryngological Society opened January 16, 1961, in Manila. It was held with the assistance of the Secretary of Health of the Philippines, Dr. Elpidio Valencia, and the Surgeon-General of the United States Air Forces, Major General O. K. Niess.

Some 200 local and foreign eye specialists and an equal number of health authorities and national and city officials attended the four-week course. The list of visiting professional lecturers included Dr. Brittain Ford Payne, Dr. Paul Chandler, Dr. Gerald Kara, Dr. Arthur Linksz, and Dr. Robb McDonald.

Temple University

The Department of Laryngology and Bronchoesophagology, Temple University Medical Center, will sponsor two postgraduate courses under the direction of Drs. Charles M. Norris and Walter H. Maloney.

Bronchoesophagology will be given September 11 to 22, 1961, and January 15 to 26, 1962. Laryngology and Laryngeal Surgery will be given April 2 to 13, 1962. The tuition fee for each course is $250. Application and further information may be obtained by writing Chevalier Jackson Clinic, Temple University Medical Center, 3401 North Broad Street, Philadelphia 40.

Aid To Cuban Ophthalmologists

The newspapers have informed the American public of the large number of Cuban refugees who have escaped the tyranny of the Castro regime in their homeland. It did not seem to be a matter that concerned us individually until it came to our attention that ten of these were ophthalmologists who had been forced to leave their practices more or less precipitately in the past several months. They were unable to bring anything out with them and for the most part are without resources.

The University of Miami School of Medicine has set up a postgraduate course to train and certify these men in order that they may qualify for work in the United States. The University, however, has no funds for the day-to-day living of these unfortunate colleagues of ours. Their need is urgent.

To meet the immediate need the Pan-American Association of Ophthalmology has sent $3000 from its treasury to the University of Miami School of Medicine who will administer all funds for the exiles. Much more is needed to help these, our colleagues in distress. It is for this reason that an urgent request for contributions is addressed to all American ophthalmologists.

Checks should be made payable to the Pan-American Association of Ophthalmology, 921 Exchange Building, Memphis 3, Tennessee.

J. Wesley McKinney, M.D.
Sec'y.-Treas. for North of Panama
Pan-American Association of
Ophthalmology

SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY
AMERICAN MEDICAL ASSOCIATION

PROGRAM

June 26-30, 1961; The Coliseum, New York, N. Y.

TUESDAY AFTERNOON, JUNE 27

Unilateral Conduction Loss in the Absence of Middle Ear Inflammation
James A. Donaldson, M.D., University Hospital, Iowa City

Address of Guest of Honor
Gordon D. Hoople, M.D., Syracuse, New York

Address of Section Chairman
Lawrence R. Boies, M.D., Minneapolis

Septal Dermoplasty
William H. Saunders, M.D., Ohio State University, Columbus, Ohio
Discussion to be opened by John Kirchner, M.D., Yale University, New Haven, Connecticut

Experiences with the Trans-Septal Trans-Sphenoidal Route in Hypophysectomy for Metastatic Carcinoma of the Breast
J. T. Noguera, M.D., and F. Robert Haase, M.D., both of 601 Grand Avenue, Asbury Park, New Jersey
Discussion to be opened by David Myers, M.D., Temple University, Philadelphia

New Concept in Rhinoplastic Procedures
Lyndon A. Peer, M.D., East Orange, New Jersey
Discussion to be opened by John Marquis Converse, M.D., New York

Safe Removal of the Nasal Hump
Irving B. Goldman, M.D., New York
Discussion to be opened by Irvin J. Fine, M.D., Perth Amboy, New Jersey, and John T. Dickinson, M.D., Pittsburgh

Results of the Treatment of Ménière's Disease with Ultrasonic Waves
Franz Altmann, M.D., and Jules G. Waltner, M.D., both of New York

BUSINESS MEETING
Report of Section Delegate and Alternate
Gordon F. Harkness, Davenport, Iowa, and Paul H. Holinger, Chicago

Report of Section Representatives to Board of Governors of the American College of Surgeons
John J. Conley, New York; Harold Owens, Los Angeles, and W. Franklin Keim, East Orange, New Jersey

Report of Nominations Committee
Victor R. Alfaro, Washington, D. C., Chairman; Francis W. Davison, Danville, Pennsylvania, and E. W. Harris, Columbus, Ohio.
Election of Officers and Nomination of Representatives to Board of Governors of the American College of Surgeons

WEDNESDAY AFTERNOON, JUNE 28

Bronchoscopy under General Anesthesia—A Simplified Safe Method
L. Q. Pang, M.D., 1374 Nuuanu Avenue, Honolulu 17
Discussion to be opened by Lyle Waggoner, M.D., Detroit and De Graaf Woodman, New York

Corrosive Esophagitis
John F. Daly, M.D., New York, and John C. Cardona, M.D., New York
Discussion to be opened by F. Johnson Putney, M.D., Philadelphia, and Daniel C. Baker, M.D., New York

Conservation of Hearing: A Clinical Analysis of Current Concepts
J. M. Cole, M.D., Danville, Pennsylvania

Clinical Application of Newer Hearing Tests
Fred Harbert, M.D., Philadelphia
Discussion to be opened by E. P. Fowler, Jr., M.D., New York, and Moe Bergman, Ph.D., Hunter College, 695 Park Avenue, New York

Secretory Otitis Media—Problems and Pitfalls
B. W. Armstrong, M.D., Charlotte, North Carolina
Discussion to be opened by Frank Lathrop, M.D., Boston, and Francis Davison, M.D., Danville, Pennsylvania

Tympanoplasty—Its Trends
Woodrow D. Schlosser, M.D., 3701 North Broad Street, Philadelphia
Discussion to be opened by Lindsay L. Pratt, M.D., Philadelphia, and Louis Silcox, M.D., Philadelphia

The Surgical Technique of Fenestration of the Oval Window (with new film)
John J. Shea, M.D., Memphis, Tennessee

Vascular Surgery in Operation on the Head and Neck
John M. Loré, Jr., M.D., Hemion Road, Suffern, New York
Discussion to be opened by John L. Madden, M.D., St. Clare's Hospital, New York, and Ralph J. Greenberg, M.D., Good Samaritan Hospital, Suffern, New York

THURSDAY, JUNE 29

OTOLARYNGOLOGY INSTRUCTION COURSES

Attendance at instruction courses is limited to fifty for each session of each course. Admission only by card, available at the meetings of the Section the previous two days; persons wishing to attend an instruction course must sign the enrollment book at the ticket table, and a ticket will be given only to the signee. These instruction courses will be of the seminar type, and must necessarily be kept to a limited attendance, therefore extra tickets will not be given to persons signing for the course, so that the tickets distributed will be definitely used. If any tickets are still available after the first two days of the Section meetings, they will then be available at the door of the instruction rooms.

1:30 to 2:30 p.m.

Room 1
Surgery of the Ear in 1961
George E. Shambaugh, Jr., M.D., Chicago

Room 2
Surgery of the Nose and Sinuses in 1961
John J. Conley, M.D., New York

Room 3
Surgery of the Throat in 1961
Joel J. Pressman, M.D., Los Angeles

2:45 to 3:45 p.m.
Second session of the above courses (all three repeated).

4:00 to 5:00 p.m.
Third session of the above courses (all three repeated).

POSITIONS

UNEXPECTED VACANCY — Approved Third Year residency in OTOLARYNGOLOGY, beginning July 1, 1961. Stipend $200.00 per month with full maintenance and training program. Apply Superintendent, Baltimore Eye, Ear, and Throat Hospital, 1214 Eutaw Place, Baltimore 17, Maryland.

WANTED—OTOLARYNGOLOGIST and OPHTHALMOLOGIST. Board certified or Board eligible for partnership practice. Town of 40,000 with a surrounding territory of 80,000 and good farming and manufacturing territory. For further information, please write L. H. Prewitt, M.D., 207 East Second Street, Ottumwa, Iowa.

WANTED—OTOLARYNGOLOGIST ASSOCIATE by Board man. Partnership later. County of 500,000. Unlimited income possibility, dependent upon ability and desire to work. Nine-room office. Average winter noon temperature of 68 degrees, giving nine wonderful months. Recreation includes fishing, golf, tennis, horses, sailing, plays, symphony orchestra, rock collecting, polo, curling, swimming and winter skiing. For further information, please write E. G. Barnet, M.D., 907 Professional Building, Phoenix 4, Arizona.

WANTED — E.E.N.T. specialist, Albuquerque, New Mexico. Population 263,000. Quality practice of recently deceased physician. Established fifteen years. Includes rental space in medical arts building, equipment, fixtures and records. Excellent opportunity for qualified specialist with special interest in contact lens field. For further information, please write Mrs. Arita H. Courville, 833 Madison, N.E., Albuquerque, New Mexico.

WANTED—Specialist in eye, ear, nose, throat and allergy work or ophthalmologist to take over practice. Unopposed practice in city of 15,000 and surrounding communities in Arizona. Equable climate. Progressive, growing community—golf, hunting and fishing. For further information, please write Louis A. Packard, M.D., Elks Building, Prescott, Arizona.

WANTED—Ear, Nose and Throat Specialist to take over active practice in Schenectady, New York. Established twenty-nine years. Completely equipped, beautifully furnished six-room office. Near downtown area and convenient to bus lines. For further information, please write Mrs. Frank C. Furlong, 713 Union Street, Schenectady, New York.

WANTED—OPHTHALMOLOGIST and OTOLARYNGOLOGIST. Board eligible or Board certified. For further information, please write Howard Trolan, M.D., 515 Soquel Avenue, Santa Cruz, California.

WANTED, OPHTHALMOLOGIST. Board qualified or certified for group practice in clinic associated with 65-bed accredited hospital in western Oklahoma. For details, write to Donald S. Lehman, Medical Director, Community Hospital, Elk City, Oklahoma.

WANTED—by a Board eligible (3-year) resident further training and experience in ocular motility. Available July 1961. For further information, please write Joseph Goldstein, M.D., 140 B Donor Avenue, East Paterson, New Jersey.

WANTED, OPHTHALMOLOGIST. Fully accredited general hospital with twenty-one doctors on staff. Community of 30,000 with surrounding area population of 59,000. Delightful climate. Very good school system and New Mexico State University. For further information, please write William L. Gold, Administrator, Memorial General Hospital, Corner Alameda and Lohman, Las Cruces, New Mexico.

WANTED, OPHTHALMOLOGIST. For 110-bed hospital in tri-city area with population of 52,000 and a drawing area population of 200,000. Completely modern hospital which is extremely well furnished for surgery. Huge plutonium project. Fast-growing agricultural economy on 200,000 acres of diversified farm land irrigated by water supplied by Grand Coulee Dam. Recreation facilities include boating, water skiing, big game hunting, and snow skiing. One of the nation's biggest flyways for migratory birds is located here with a plentiful supply of upland birds. For further information, please write to Richard Lubben, Administrator, Kadlec Methodist Hospital, Richland, Washington.

FOR SALE

OPHTHALMOLOGIST'S Active, Large Practice and office equipment of prominent Ophthalmologist who died recently of sudden illness. Records available. Will be released only to an accredited or Board eligible Ophthalmologist. REPLY IN WRITING TO Mrs. Carroll R. Mullen, 2025 Locust Street, Philadelphia 3, Pennsylvania.

Listings of positions available should be sent to W. L. Benedict, M.D., 15 Second Street S. W., Rochester, Minn., by the fifth of the month preceding publication. They should include (1) type of physician wanted (ophthalmologist or otolaryngologist: diplomate, resident, etc.) (2) type of position to be filled; and (3) to whom to write for further information. Unless otherwise requested, listings will be published only once.

The Academy will handle no further correspondence beyond the listing and assume no responsibility. Neither does it endorse nor guarantee any of the published listings.

DIRECTORY OF OPHTHALMIC AND OTOLARYNGIC SOCIETIES

Secretaries of Societies are requested to furnish the information necessary to make this list complete and keep it up to date. Send to TRANSACTIONS, *American Academy of Ophthalmology and Otolaryngology,* 15 *Second St. S.W., Rochester, Minn.*

EXAMINING AND CERTIFYING BODIES

AMERICAN BOARD OF OPHTHALMOLOGY

CHAIRMAN: Kenneth C. Swan, M.D.
SECRETARY-TREASURER: Dr. Merrill J. King, Ivie Rd., Box 236, Cape Cottage Branch, Portland 9, Maine
ORAL EXAMINATION: October 2-6, 1961, Chicago
FINAL DATE FOR APPLICATION TO TAKE 1961 WRITTEN EXAMINATION: July 1, 1961
WRITTEN EXAMINATION: January 22, 1962

AMERICAN BOARD OF OTOLARYNGOLOGY

PRESIDENT: Bernard J. McMahon, M.D.
SECRETARY-TREASURER: Dr. Dean M. Lierle, University Hospitals, Iowa City
ORAL EXAMINATION: October 2-6, 1961, Chicago
FINAL DATE FOR APPLICATION: March 31, 1961

AMERICAN BOARD OF PLASTIC SURGERY

CHAIRMAN: Dr. James T. Mills
SECRETARY-TREASURER: Dr. Bradford Cannon, 330 Dartmouth St., Boston 16
CORRESPONDING SECRETARY: Estelle E. Hillerich, 4647 Pershing Ave., St. Louis 8
FINAL DATE FOR SUBMITTING CASE REPORTS: January 1, 1961
ORAL AND WRITTEN EXAMINATIONS: May 14-16, 1961; New York City

THE ROYAL COLLEGE OF PHYSICIANS AND SURGEONS OF CANADA

PRESIDENT: Donald A. Thompson, F.R.C.S. (C)
HONORARY SECRETARY: Dr. James H. Graham, 150 Metcalfe St., Ottawa 4, Ont.
TIME AND PLACE: January 18-20, 1962, Royal York Hotel, Toronto, Ont., Canada

INTERNATIONAL

ASIA-PACIFIC ACADEMY OF OPHTHALMOLOGY

PRESIDENT: Dr. Geminiano de Ocampo
SECRETARY-GENERAL: Dr. William John Holmes, 280 Young Hotel Bldg., Honolulu 13, Hawaii
TIME AND PLACE: December 3, 1962, New Delhi, India

EUROPEAN OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Prof. G. von Bahr
SECRETARY: Prof. Jules Francois, 15 Place de Smet de Naeyer, Ghent, Belgium
TIME AND PLACE: 1964; Vienna, Austria

INTERNATIONAL ASSOCIATION FOR THE PREVENTION OF BLINDNESS

PRESIDENT: Prof. A. Franceschetti
SECRETARY: Dr. J. P. Bailliart, 47 rue de Bellechasse, Paris (7e), France
TIME AND PLACE: December 1962; New Delhi

INTERNATIONAL ASSOCIATION OF SECRETARIES OF OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETIES

PRESIDENT: Peter N. Pastore, M.D.
SECRETARY: Dr. J. David Stratton, 1012 Kings Drive, Charlotte, N. C.
TIME AND PLACE: October 10, 1961; Palmer House, Chicago

INTERNATIONAL BRONCHOESOPHAGOLOGICAL SOCIETY

PRESIDENT: Prof. Giorgio Ferreri
SECRETARY: Dr. Chevalier L. Jackson (deceased)
TIME AND PLACE: July, 1961; 10th International Congress of Bronchoesophagology, Dusseldorf, Reims, Paris

INTERNATIONAL COLLEGE OF SURGEONS, SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Dr. Clarence H. Albaugh
SECRETARY: Dr. Louis Savitt, 3403 Lawrence Ave., Chicago 25
TIME AND PLACE: May 7-11, 1961; Palmer House, Chicago

INTERNATIONAL FEDERATION OF OPHTHALMOLOGICAL SOCIETIES

PRESIDENT: Sir Stewart Duke-Elder
SECRETARY: Dr. Edward Hartmann, 2 Ave. Ingres, Paris (16e), France
TIME AND PLACE: December 3-7, 1962; New Delhi

INTERNATIONAL ORGANIZATION AGAINST TRACHOMA

PRESIDENT: Prof. G. B. Bietti
SECRETARY-GENERAL: Dr. Jean Sedan, 94 rue Sylvabelle, Marseille (B. du R.), France
TIME AND PLACE: December 1962; New Delhi

PAN-AMERICAN ASSOCIATION OF OPHTHALMOLOGY

PRESIDENT: Jorge Valdeavellano, M.D.
SECRETARY-TREASURER: For North of Panama: Dr. J. Wesley McKinney, 921 Exchange Bldg., Memphis
For South of Panama: Renato de Toledo, Sao Paulo, Brazil
TIME AND PLACE: February, 1962, Lima Peru and October 4-9, 1964, Montreal, Canada

PAN-AMERICAN ASSOCIATION OF
OTO-RHINO-LARYNGOLOGY AND BRONCHO-ESOPHAGOLOGY

PRESIDENT: Dr. Plinio Barretto
SECRETARY: Dr. Chevalier L. Jackson (deceased)
TIME AND PLACE: 1962; Caracas, Venezuela

FOREIGN

Argentina

SOCIEDAD ARGENTINA DE OFTALMOLOGIA

PRESIDENT: Dr. Paulina Satanowsky de Neumann
SECRETARY: Dr. Atilio L. Norbis, Santa Fé 1171 Capital Federal
TIME AND PLACE: Third Wednesday of each month; Santa Fé 1171, Buenos Aires

SOCIEDAD de OFTALMOLOGIA de CORDOBA

PRESIDENT: Prof. Dr. Alberto Urrets-Zavalia, Jr., Casilla Correo 301, Cordoba, Argentina
SECRETARY: Dr. Claudio Juarez Beltran
SECRETARY-TREASURER: Dr. Luis Mitnik
MEMBERSHIP: Dr. Juan Remonda and Dr. Dante Yadarola

Australia

OPHTHALMOLOGICAL SOCIETY OF AUSTRALIA (B.M.A.)

PRESIDENT: Dr. T. a'B. Travers
SECRETARY: Dr. John K. Hart, 27 Commonwealth St., Sydney
TIME AND PLACE: October 9-13, 1961; Canberra

Austria

OSTERREICHISCHE OPHTHALMOLOGISCHE GESELLSCHAFT

PRESIDENT: Prof. Dr. K. Hruby
SECRETARY: Dr. Franz Schwab, I. Univ. Augenklinik, Alserstr. 4, Vienna 9

OSTERREICHISCHE OTO-LARYNGOLOGISCHE GESELLSCHAFT

PRESIDENT: Prof. Dr. Otto Novotny
SECRETARY: Dr. Ludwig Bablik, Vienna IX, Alserstrasse 4
TIME: First Tuesday of each month; October to June

OPHTHALMOLOGISCHE GESELLSCHAFT IN WIEN

PRESIDENT: Dozent Dr. F. A. Hamburger
SECRETARY: Dr. H. Schenk, I. Univ. Augenklinik, Alserstr. 4, Vienna 9
TIME AND PLACE: Second Monday—January through December, 2 Univ. Augenklinik, Alserstr. 4, Vienna

Belgium

SOCIETE BELGE D'OPHTALMOLOGIE

SECRETARY: Prof. Dr. M. Applemans, 179 Avenue Ruelens, Louvain

Brazil

CENTRO DE ESTUDOS DE OFTALMOLOGIA

PRESIDENT: Dr. Armando de Arruda Novaes
SECRETARY: Dr. Jose Carlos Gouvea Pacheco, R. Cons. Crispinn'ano, 53-8, Sao Paulo
TIME AND PLACE: Every Tuesday; R. Condessa de S. Joaquim 288, Sao Paulo

Canada

CANADIAN MEDICAL ASSOCIATION, SECTION OF OPHTHALMOLOGY

CHAIRMAN: Dr. Edward F. Foy
SECRETARY: Dr. Robert W. Robertson, 305 Northgate Bldg., Edmonton, Alta.

CANADIAN OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Dr. B. Alexander
SECRETARY: Dr. R. G. C. Kelly, 90 St. Clair Ave. W., Toronto 7, Ont.
TIME AND PLACE: June 11-14, 1961; Queen Elizabeth Hospital, Montreal, Canada

CANADIAN OTOLARYNGOLOGICAL SOCIETY

PRESIDENT: Fernand Montreuil, M.D.
SECRETARY: Donald M. MacRae, M.D., 324 Spring Garden Rd., Halifax, N. S.
TIME AND PLACE: June 15-17, 1961; Montreal

OPHTHALMOLOGIC AND OTOLARYNGOLOGIC SOCIETY OF ALBERTA

PRESIDENT: M. G. Macaulay, M.D.
SECRETARY: M. R. Marshall, M.D., North Pavilion, University Hospital, Edmonton, Alta.
TIME AND PLACE: April, 1961; Edmonton

NOVA SCOTIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: H. J. Davidson, M.D.
SECRETARY: C. F. Keays, M.D., 324 Spring Garden Rd., Halifax, N. S.

THE MONTREAL MEDICO-CHIRURGICAL SOCIETY, SECTION ON OTOLARYNGOLOGY

PRESIDENT: Dr. H. E. McHugh
SECRETARY: Dr. Paul Ittkin, 600 Medical Arts Bldg., Montreal 25, Que.
TIME AND PLACE: November, January, February and April; Teaching Hospitals

MONTREAL OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Dr. John C. Locke
SECRETARY: Dr. J. Noiseux, 400 Sherbrooke Street East

TORONTO ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY

PRESIDENT: Dr. Lois A. Lloyd
SECRETARY: Dr. John W. Hiltz, 741 Bayview Ave., Toronto, Ont.

Chile

SOCIEDAD CHILENA DE OFTALMOLOGIA

PRESIDENT: Dr. Juan Verdaguer
SECRETARY: Dr. Wolfran Rojas, Casilla 13017 Providencia, Santiago

Colombia

SOCIEDAD COLOMBIANA DE OFTALMOLOGIA Y OTO-RINO-LARINGOLOGIA

PRESIDENT: Dr. Alfonso Gaitan Nieto
SECRETARY: Dr. Juan Arciniegas, Calle 47 #5-26, Bogotá
TIME AND PLACE: Monthly; Bogotá

Cuba

SOCIEDAD CUBANA DE OFTALMOLOGIA

PRESIDENT: Dr. Manuel Iglesias Revuelta
SECRETARY: Dr. Jaime Alemany Martorell, 23 #301 esq. a L., Vedado, LaHabana
TIME: Annual in December

Denmark

DET OFTALMOLOGISKE SELSKAB I KØBENHAVN

PRESIDENT: dr. med. Henning Skydsgaard
SECRETARY: dr. med. A. Mahneke, University Hospital, Eye Department, Copenhagen

France

SOCIETE FRANCAISE D'OPHTALMOLOGIE

PRESIDENT: Prof. Guy Offret
SECRETARY: Dr. Gabriel Lombard, 27 rue du Faubourg, St. Jacques, Paris
TIME AND PLACE: Maison de la Chimie, 28 rue Saint Dominique, Paris

SOCIETE FRANCAISE D'OTO-RHINO-LARYNGOLOGIE

PRESIDENT: Prof. André Soulas
SECRETARY: Dr. Henry Guillon, 6 Av. MacMahon, Paris

Germany

DEUTSCHE OPHTHALMOLOGISCHE GESELLSCHAFT

PRESIDENT: Prof. Dr. W. Rohrschneider
SECRETARY: Prof. Dr. W. Jaeger, Univ. Augenklinik, Heidelberg
TIME AND PLACE: September 24-28, 1961, Heidelberg

Great Britain

BRITISH ASSOCIATION OF OTOLARYNGOLOGISTS

PRESIDENT: Mr. V. F. Lambert
SECRETARY: Mr. G. H. Bateman, 47 Lincoln's Inn Fields, London, W. C. 2

FACULTY OF OPHTHALMOLOGISTS

PRESIDENT: Mr. E. G. Mackie
SECRETARY: Mr. J. R. Hudson, 47 Lincoln's Inn Fields, London, W. C. 2

MIDLAND OPHTHALMOLOGICAL SOCIETY

PRESIDENT: C. H. Bamford
SECRETARIES: W. Martin Walker and A. N. Cameron, 51 Calthorpe Road, Edgbaston, Birmingham 15, England
TIME AND PLACE: September 22, 1961; Annual Meeting

OPHTHALMOLOGICAL SOCIETY OF THE UNITED KINGDOM

PRESIDENT: Mr. Frank W. Law
SECRETARIES: Mr. L. G. Fison and Mr. Redmond Smith, 47 Lincoln's Inn Fields, London W. C. 2

OXFORD OPHTHALMOLOGICAL CONGRESS

MASTER: A. B. Nutt
SECRETARY: Mr. Ian C. Fraser, 21 Dogpole, Shrewsbury, Shropshire
TIME AND PLACE: July 10-12, 1961; University Laboratory of Physiology, South Parks Rd., Oxford

THE ROYAL SOCIETY OF MEDICINE, SECTION ON OPHTHALMOLOGY

PRESIDENT: Sir Benjamin Rycroft
SECRETARIES: W. E. S. Bain and Barrie R. Jones, 1 Wimpole Street, London, W.1.
TIME AND PLACE: Second Thursday, October through December, February through June; 1 Wimpole St., London, W. 1.

THE ROYAL SOCIETY OF MEDICINE, SECTION ON OTOLOGY

PRESIDENT: Mr. J. P. Monkhouse
SECRETARY: Mr. E. F. Stewart, 33 Alwgue Road, Canonbury London N.1
TIME AND PLACE: First Friday, November through June, except January and April; 1 Wimpole St., London W. 1.

THE ROYAL SOCIETY OF MEDICINE, SECTION ON LARYNGOLOGY

PRESIDENT: J. H. Otty, F.R.C.S. Ed.
SECRETARIES: E. H. Miles Foxen, F.R.C.S.
Alun B. Thomas, F.R.C.S., 1 Wimpole Street, London, W. 1
TIME AND PLACE: First Friday of the month, November through May

SCOTTISH OPHTHALMOLOGICAL CLUB

PRESIDENT: Dr. J. R. Paterson
SECRETARIES: Dr. C. R. S. Jackson, 1 Melville St., Edinburgh 3
Dr. D. Christison, 21 Eastcote Ave., Glasgow, W. 4

SOUTHERN OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Mrs. Janet Simpson, M.B., Ch.B., D.O.
SECRETARY: Nigel Cridland, D.M., D.O.

India

ALL-INDIA OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Dr. Tulsidas
SECRETARY: Dr. Y. K. C. Pandit, Bombay Mutual Building, Fort, Bombay

BOMBAY OPHTHALMOLOGISTS' ASSOCIATION

PRESIDENT: H. D. Dastoor
SECRETARY: Dr. T. N. Ursekar, T. N. M. College, Nair Road, Byculla, Bombay 8
TIME AND PLACE: 7:30 p.m., Third Wednesday of each month; various hospitals

THE OPHTHALMOLOGICAL SOCIETY OF BENGAL

PRESIDENT: N. K. Munsi, M.D.
JOINT SECRETARIES: A. K. Sen, M.B., D.O.M.S., and S. Chatterjee, M.B., F.R.C.S.
TIME AND PLACE: Medical College Hospitals, Eye Infirmary, Calcutta, India

Ireland

IRISH OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Dr. L. B. Somerville-Large
SECRETARY: Dr. T. F. Roche, 25 FitzWilliam Pl., Dublin
TIME AND PLACE: April 27-29, 1961; Dublin

Italy

SOCIETA OFTALMOLOGICA ITALIANA

PRESIDENT: Prof. Luigi Maggiore
SECRETARY: Prof. E. Leonardi, Piazza Degli Eroi 11, Roma

Mexico

SOCIEDAD MEXICANA de OFTALMOLOGIA

PRESIDENT: Dr. Renan Murillo Fajardo
SECRETARY: Dr. Sabino Silva Zeron, Brasil 33, Mexico 1, D. F.

The Netherlands

NEDERLANDSCH OOGHEELKUNDIG GEZELSCHAP

PRESIDENT: Prof. Dr. M. C. Colenbrander
SECRETARY: Dr. P. G. Binkhorst, Sweelincklaan 20, Bilthoven

New Zealand

THE OPHTHALMOLOGICAL SOCIETY OF NEW ZEALAND

PRESIDENT: Dr. Lindsay Burns
SECRETARY: Dr. Randal Elliott, 89 The Terrace, Wellington C1, N.Z
TIME AND PLACE: October 16-20, 1961; Wairakei, North Island, New Zealand

Norway

NORSK OFTALMOLOGISK FORENING

PRESIDENT: Dr. Einar Trumpy
SECRETARY: Otto Johansen, Teatergarten 11, Oslo
TIME AND PLACE: Four times a year; Rikshospitalets Øyeavdeling, Oslo

Pakistan

OPHTHALMOLOGICAL SOCIETY OF PAKISTAN

PRESIDENT: Lt. Gen. S. M. A. Farugi
SECRETARY: Dr. Raja Mumtaz, 70 Nedous Hotel, Lahore
TIME AND PLACE: K. E. Medical College, Lahore

Paraguay

SOCIETY OF OPHTHALMOLOGY OF PARAGUAY

PRESIDENT: Dr. Honorio Campuzano
SECRETARY: Emilio Gonzales Gini, Estados Unidos 183 Asunción, Paraguay
TIME AND PLACE: Monthly; Circulo Paraguayo de Medicos, Asunción

Philippine Islands

PHILIPPINE SOCIETY OF OTOLARYNGOLOGY AND BRONCHOESOPHAGOLOGY

PRESIDENT: Cesar F. Villafuerte, M.D.
SECRETARY-TREASURER: Eusebio E. Llamas, M.D., 492 Padre Faura Street, Manila, Philippines

Spain

INSTITUTO BARRAQUER

PRESIDENT: Ignacio Barraquer, M.D.
SECRETARY: Joaquin Rutllan, M.D., Instituto Barraquer, Laforja 88, Barcelona, Spain

Sweden

SVENSKA OGONLAKAREFORENINGEN,
SVENSKA LAKARESALLSKAPETS SEKTION FOR OFTALMOLOGI

PRESIDENT: Prof. B. Rosengren
SECRETARY: Dr. Ilmari Rendahl, Eye Clinic Karolinska Sjukhuset, Stockholm 60
TIME AND PLACE: June 1961; Gothenburg

Union of South Africa

THE OPHTHALMOLOGICAL SOCIETY OF SOUTH AFRICA

PRESIDENT: Dr. H. J. Hamelberg
SECRETARY: Dr. E. T. Meyer, 220 Lister Bldg., Jeppe St., Johannesburg

Venezuela

SOCIEDAD VENEZOLANA DE OTORINOLARINGOLOGIA

PRESIDENT: Dr. Oscar Bustamante Miranda
SECRETARY: Dr. R. Marquez Landaeta, Colegio de Medicos, Los Chaguaramos Caracas, Venezuela
TIME AND PLACE: November 16-19, 1961; Valencia, Edo. Carabobo

NATIONAL

AMERICAN ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Dohrmann K. Pischel, M.D.
EXECUTIVE SECRETARY-TREASURER: William L. Benedict, M.D., 15 Second St. S.W., Rochester, Minn.
TIME AND PLACE: October 8 to 13, 1961; Palmer House, Chicago

AMERICAN BRONCHO-ESOPHAGOLOGICAL ASSOCIATION

PRESIDENT: F. Johnson Putney, M.D.
SECRETARY: Daniel C. Baker, Jr., M.D., 903 Park Avenue, New York 21
TIME AND PLACE: May 23 and 24, 1961; Lake Placid Club, Lake Placid, N. Y.

AMERICAN LARYNGOLOGICAL ASSOCIATION

PRESIDENT: Edwin N. Broyles, M.D.
SECRETARY: Lyman G. Richards, M.D., 12 Clovelly Road, Wellesley Hills 81, Mass.
TIME AND PLACE: May 21 and 22, 1961; Lake Placid Club, Lake Placid, N. Y.

AMERICAN LARYNGOLOGICAL, RHINOLOGICAL AND OTOLOGICAL SOCIETY, INC.

PRESIDENT: Fletcher D. Woodward, M.D.
SECRETARY: C. Stewart Nash, M.D., 708 Medical Arts Bldg., Rochester 7, N. Y.
TIME AND PLACE: May 23-25, 1961; Lake Placid Club, Lake Placid, N. Y.

EASTERN SECTION

CHAIRMAN: Frank D. Lathrop, M.D.
SECRETARY: Alfred W. Doust, M.D., State Tower Bldg., Syracuse, N. Y.

SOUTHERN SECTION

CHAIRMAN: Peter N. Pastore, M.D.
SECRETARY: James A. Harrill, M.D., Bowman Gray School of Medicine, Winston-Salem, N. C.

MIDDLE SECTION

SECRETARY: Myron Hipskind, M.D., Suite 3000, 55 East Washington Street, Chicago

WESTERN SECTION

CHAIRMAN: Joel J. Pressman, M.D.
SECRETARY: Francis A. Sooy, M.D., 490 Post Street, San Francisco

AMERICAN MEDICAL ASSOCIATION, SECTION ON LARYNGOLOGY, OTOLOGY AND RHINOLOGY

CHAIRMAN: Lawrence R. Boies, M.D.
SECRETARY: Walter E. Heck, M.D., Stanford Hospital, San Francisco 15
TIME AND PLACE: June 26-30, 1961; New York

AMERICAN MEDICAL ASSOCIATION, SECTION ON OPHTHALMOLOGY

CHAIRMAN: Banks Anderson, M.D.
SECRETARY: Henry F. Allen, M.D., 200 Beacon St., Boston 16, Mass.
TIME AND PLACE: June 26-30, 1961; New York City

AMERICAN OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Edwin B. Dunphy, M.D.
SECRETARY-TREASURER: Joseph A. C. Wadsworth, M.D., 108 East 68th Street, New York 21

AMERICAN OTOLOGICAL SOCIETY, INC.

PRESIDENT: Henry L. Williams, M.D.
SECRETARY-TREASURER: James A. Moore, M.D., 525 East 68th Street, New York 21
TIME AND PLACE: May 26 and 27, 1961; Lake Placid Club, Lake Placid, N. Y.

THE AMERICAN OTORHINOLOGIC SOCIETY FOR PLASTIC SURGERY, INC.

PRESIDENT: Louis J. Feit, M.D.
SECRETARY: Joseph G. Gilbert, M.D., 75 Barberry Lane, Roslyn Heights, N. Y.

AMERICAN SOCIETY OF FACIAL PLASTIC SURGERY

PRESIDENT: Sam H. Sanders, M.D.
SECRETARY: Samuel M. Bloom, M.D., 123 E. 83rd St., New York 28
TIME AND PLACE: June 28, 1961; Hotel Elysee, New York City

AMERICAN SOCIETY FOR HEAD AND NECK SURGERY

PRESIDENT: John J. Conley, M.D.
SECRETARY: George A. Sisson, M.D., 1200 East Genesee Street, Syracuse 10
TIME AND PLACE: October 7, 1961; Palmer House, Chicago

AMERICAN SOCIETY OF OPHTHALMOLOGIC AND OTOLARYNGOLOGIC ALLERGY

PRESIDENT: Leland H. Prewitt, M.D.
SECRETARY: Daniel S. De Stio, M.D., 121 South Highland Avenue, Pittsburgh 6
TIME AND PLACE: October 7, 1961; Sherman Hotel, Chicago

ASSOCIATION FOR RESEARCH IN OPHTHALMOLOGY, INC.

CHAIRMAN: V. Everett Kinsey, Ph.D.
SECRETARY: Monte G. Holland, M.D., 1430 Tulane Avenue, New Orleans 12
TIME AND PLACE: June 26-28, 1961; Plaza Hotel, New York

NATIONAL SOCIETY FOR THE PREVENTION OF BLINDNESS

PRESIDENT: Mr. Enos Curtin
EXECUTIVE DIRECTOR: John W. Ferree, M.D.
SECRETARY: Regina E. Schneider, 1790 Broadway, New York 19
TIME AND PLACE: April 12-14, 1961; Barbizon Plaza Hotel, New York City

NATIONAL MEDICAL FOUNDATION FOR EYE CARE

PRESIDENT: Ralph O. Rychener, M.D.
SECRETARY: Charles E. Jaeckle, M.D., 250 West 57th Street, New York 19
TIME AND PLACE: October 8, 1961; Chicago

OTOSCLEROSIS STUDY GROUP

PRESIDENT: Arthur L. Juers, M.D.
SECRETARY: Raymond Jordan, M.D., 3515 Fifth Ave., Pittsburgh
TIME AND PLACE: October 7 and 8, 1961, Palmer House, Chicago.

PUERTO RICAN OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Manuel N. Miranda, M.D.
SECRETARY: Guillermo Picó, M.D., 1475 Wilson Ave., Santurce
TIME AND PLACE: Bimonthly; Puerto Rican Medical Association Bldg.

SOCIETY OF MILITARY OPHTHALMOLOGISTS

PRESIDENT: Aubrey Jennings, M.D.
SECRETARY: Capt. Don W. Longfellow, MC, USA, Armed Forces Institute of Pathology, WRAMC, Washington 12, D. C.

SOCIETY OF MILITARY OTOLARYNGOLOGISTS

PRESIDENT: Col. James E. Lett, USAF MC
SECRETARY-TREASURER: Capt. G. W. Hurst, MC, USN, U. S. Naval Hospital, Great Lakes, Ill.
TIME AND PLACE: October, 1961; Chicago

REGIONAL

ARK-LA-TEX OTO-OPHTHALMIC SOCIETY

PRESIDENT: Jack W. Pou, M.D.
SECRETARY: Richard Gibson, M.D., Doctors Bldg., Margaret Place, Shreveport, La.
TIME AND PLACE: 7:00 p.m., First Monday of each month, September through June; Confederate Memorial Hospital, Shreveport, La.

LOUISIANA-MISSISSIPPI OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

PRESIDENT: Arthur V. Hays, M.D.
SECRETARY: Edley H. Jones, M.D., 1301 Washington St., Vicksburg, Miss.
TIME AND PLACE: May 12 and 13, 1961; Edgewater Gulf Hotel, Edgewater Park, Miss.

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

PRESIDENT: David G. Cogan, M.D.
SECRETARY: Alfred W. Scott, M.D., 7 Bay State Road, Boston 15, Mass.
TIME AND PLACE: Third Wednesday, November through April; Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston

NEW ENGLAND OTO-LARYNGOLOGICAL SOCIETY

PRESIDENT: Frank Turchik, M.D.
SECRETARY: Burton E. Lovesey, M.D., 15 W. Central St., Natick, Mass.
PLACE: Massachusetts Eye and Ear Infirmary, 243 Charles St., Boston

PACIFIC COAST OTO-OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Dohrmann K. Pischel, M.D.
SECRETARY: Alden H. Miller, M.D., 500 South Lucas Avenue, Los Angeles 17
TIME AND PLACE: April, 1961; Palm Springs, Calif.

SIOUX VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: J. Calvin Davis, M.D.
SECRETARY: Verne R. Heimann, M.D., 507 7th Street, Sioux City, Iowa

SOUTHERN MEDICAL ASSOCIATION SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Miles L. Lewis, Jr., M.D.
SECRETARY: Dr. Albert C. Esposito, First Huntington National Bank Bldg., Huntington, W. Va.
TIME AND PLACE: November, 1961; Dallas

WISCONSIN-UPPER MICHIGAN SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: W. J. Troup, M.D.
SECRETARY: G. L. McCormick, M.D., 630 S. Central Ave., Marshfield, Wis.
TIME AND PLACE: April 15 and 16, 1961; Wausau, Wisconsin

STATE AND LOCAL

Alabama

ALABAMA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: John Allen Jones, M.D.
SECRETARY: Karl B. Benkwith, M.D., 14 Catoma St., Montgomery

THE BIRMINGHAM EYE, EAR, NOSE AND THROAT SOCIETY

PRESIDENT: Each member in alphabetical order
SECRETARY: Joseph M. Dixon, M.D., 101 Medical Arts Bldg., Birmingham 5
TIME AND PLACE: Monthly; The Club

Arizona

PHOENIX OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Virgil A. Toland, M.D.
SECRETARY: Roy E. Burgess, M.D., 461 West Catalina Drive, Phoenix
TIME AND PLACE: 6:30 p.m., Third Thursday every other month; Westward Ho Hotel

Arkansas

ARKANSAS MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

PRESIDENT: M. E. Blanton, M.D.
SECRETARY: James L. Smith, M.D., 623 Woodlane, Little Rock
TIME AND PLACE: May, 1961; Little Rock

California

CALIFORNIA MEDICAL ASSOCIATION, EYE SECTION

PRESIDENT: Earle H. McBain, M.D.
SECRETARY: Floyd M. Bond, M.D., 625 Broadway, San Diego

CALIFORNIA MEDICAL ASSOCIATION, EAR, NOSE AND THROAT SECTION

PRESIDENT: H. W. Kohlmoos, M.D.
SECRETARY: Marvin Simmons, M.D., Box 4003, Fresno
TIME AND PLACE: May 1, 1961; Los Angeles

ALTA CALIFORNIA OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Arthur R. Kahler, M.D.
SECRETARY: Julian J. Fisher, M.D., 2600 Capitol Avenue, Sacramento

EAST BAY OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Ford Keppen, M.D.
SECRETARY: Lionel Sorenson, M.D., 2320 Channing Way, Berkeley, Calif.

LOS ANGELES OPHTHALMOLOGICAL JOURNAL CLUB

PRESIDENT: Henry Montross, M.D.
SECRETARY: Jacob M. Kline, M.D., 4070 Buckingham Road, Los Angeles 8
TIME AND PLACE: 8:00 p.m., Second Monday of each month, Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles

LOS ANGELES SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Herschel H. Burston, M.D.
SECRETARY: Richard Kratz, M.D., 15225 Van Owen, Van Nuys
TIME AND PLACE: Ophthalmology, 6:00 p.m., First Thursday of each month;
Otolaryngology, 6:00 p.m., Fourth Monday of each month;
Los Angeles County Medical Association Bldg., 1925 Wilshire Blvd., Los Angeles

PENINSULA EYE SOCIETY

PRESIDENT: Robert D. Cook, M.D.
SECRETARY: Frank D. Berry, M.D., 762 Altos Oaks Drive, Los Altos, Calif.
TIME: Third Thursday evening of each month

SAN DIEGO OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Martin P. Koke, M.D.
SECRETARY: Cody L. Smith, M.D., 125 Prescott Avenue, El Cajon, Calif.
TIME AND PLACE: First Monday of each month, Mission Valley Inn

SAN FRANCISCO MEDICAL SOCIETY, EYE SECTION

CHAIRMAN: Alan J. Rosenberg, M.D., 450 Sutter Street, San Francisco
TIME AND PLACE: Quarterly; San Francisco County Medical Bldg.

SAN FRANCISCO OPHTHALMOLOGICAL ROUND TABLE

PRESIDENT: O. Ralph Tanner, M.D.
SECRETARY-TREASURER: Albert J. Brinckerhoff, M.D., 490 Post St., San Francisco 2

SANTA BARBARA OPHTHALMOLOGICAL SOCIETY

CHAIRMAN: Harold B. Alexander, M.D.
SECRETARY: F. J. Kanofsky, M.D., 1734 State Street, Santa Barbara, Calif.
TIME AND PLACE: 7:30 p.m., Fourth Thursday evening of each month, St. Francis Hospital Library

TRI-COUNTY SOCIETY OF OPHTHALMOLOGY

PRESIDENT: Louis George, M.D.
SECRETARY: Jerry Donin, M.D., 1129 North Garey Avenue, Pomona, Calif.

Colorado

COLORADO OPHTHALMOLOGICAL SOCIETY

PRESIDENT: William H. Droegemueller, M.D.
SECRETARY: Max Kaplan, M.D., 216 Republic Bldg., Denver 2
TIME AND PLACE: Third Saturday of each month, September through May; University of Colorado Medical Center, 4200 E. Ninth Ave., Denver

COLORADO OTOLARYNGOLOGICAL SOCIETY

PRESIDENT: Sidney Fieman, M.D.
SECRETARY: Harland Close, M.D., Veteran's Administration Hospital, Denver
TIME AND PLACE: First Saturday of each month; Denver County Medical Library

Connecticut

CONNECTICUT STATE MEDICAL SOCIETY, EYE, EAR, NOSE AND THROAT SECTION

PRESIDENT: Charles J. Petrillo, M.D.
SECRETARY: Arthur C. Unsworth, M.D., 85 Jefferson Street, Hartford 14

District of Columbia

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON OPHTHALMOLOGY

PRESIDENT: Elbert W. Dodd, Jr., M.D.
SECRETARY-TREASURER: Henry L. Bastien, M.D., 3801 Fairfax Drive, Arlington

MEDICAL SOCIETY OF THE DISTRICT OF COLUMBIA, SECTION ON OTOLARYNGOLOGY

PRESIDENT: Max Fischer, M.D.
SECRETARY: Robert D. Ralph, M.D., 6220 North Washington Blvd., Arlington, Va.
TIME AND PLACE: 6:30 p.m., Second Tuesday of November, January, March and May; Army Navy Club

Florida

FLORIDA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Kenneth S. Whitmer, M.D.
SECRETARY: Joseph Taylor, Jr., M.D., Suite 709, One Davis Blvd., Tampa
TIME AND PLACE: April, 1961; The Americana, Miami Beach

GREATER MIAMI EYE, EAR, NOSE AND THROAT SOCIETY

PRESIDENT: Mariano C. Caballero, M.D.
SECRETARY-TREASURER: H. Carlton Howard, M.D., Suite 219, Plaza Bldg., 245 S. E. First St., Miami 32
TIME AND PLACE: Quarterly; McAllister Hotel

Georgia

GEORGIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: W. P. Rhyne, M.D.
SECRETARY: James T. King, M.D., 516 Baptist Professional Bldg., Atlanta 12
TIME AND PLACE: March 2-4, 1961; General Oglethorpe Hotel, Wilmington Island, Savannah, Ga.

ATLANTA EYE, EAR, NOSE AND THROAT SOCIETY

PRESIDENT: Lester A. Brown, M.D.
SECRETARY: Harry Arnold, M.D., 1293 Peachtree N. E.
TIME AND PLACE: 7:30 p.m., Fourth Monday of each month; Fulton County Academy of Medicine

Hawaii

HAWAII EYE, EAR, NOSE AND THROAT SOCIETY

PRESIDENT: W. T. Minatoya, M.D.
SECRETARY: Herbert G. Pang, M.D., Suite 202, Nuuanu Medical Center, 1374 Nuuanu Avenue, Honolulu 17, Hawaii
TREASURER: Harold T. Kimata, M.D.
TIME AND PLACE: Third Thursday of each month; Pacific Club

Illinois

CENTRAL ILLINOIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Dr. G. E. Hartenbower
SECRETARY: Dr. C. L. Pannabecker, 434 Jefferson Bldg., Peoria, Ill.

CHICAGO LARYNGOLOGICAL AND OTOLOGICAL SOCIETY

PRESIDENT: Henry B. Perlman, M.D.
SECRETARY-TREASURER: Robert Lewy, M.D., 25 E. Washington St., Chicago 2
TIME AND PLACE: First Monday of each month, October through May; Furniture Club of America, 666 N. Lake Shore Dr., Chicago

CHICAGO OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Joseph Haas, M.D.
SECRETARY: David Shoch, M.D., 700 North Michigan Ave., Chicago 11
TIME AND PLACE: Third Monday of each month, October to May; Illini Union, 715 S. Wood St., Chicago

Indiana

INDIANA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Ernest L. Dietl, M.D.
SECRETARY: Carlton A. Keck, M.D., 2902 Fairfield Avenue, Fort Wayne
TIME AND PLACE: May 4 and 5, 1961; French Lick Sheraton Hotel, French Lick, Ind.

INDIANAPOLIS OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

PRESIDENT: David E. Brown, M.D.
SECRETARY: Theodore R. LeMaster, M.D., 305 Hume Mansur Bldg., Indianapolis
TIME AND PLACE: 6:30 p.m., Second Thursday of each month, November through May; Indianapolis Athletic Club

Iowa

IOWA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Chester C. Woodburn, Jr., M.D.
SECRETARY: William Huffman, M.D., University Hospitals, Iowa City
TIME AND PLACE: September 15 and 16, 1961; Lake Okoboji, Iowa

DES MOINES ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Robert H. Foss, M.D.
SECRETARY: J. H. McNamee, M.D., 306 Equitable Bldg., Des Moines 9, Iowa
TIME AND PLACE: Quarterly: last Monday of the month; Des Moines Club

Kentucky

LOUISVILLE EYE AND EAR SOCIETY

PRESIDENT: Albert Leggett, Jr., M.D.
SECRETARY: Roderick MacDonald, Jr., M.D., 323 East Chestnut Street, Louisville
TIME AND PLACE: Bimonthly; Provincial House

Louisiana

NEW ORLEANS GRADUATE MEDICAL ASSEMBLY

PRESIDENT: Maurice E. St. Martin, M.D.
SECRETARY: Mannie D. Paine, Jr., M.D., Room 103, 1430 Tulane Ave., New Orleans

Maryland

BALTIMORE CITY MEDICAL SOCIETY, OPHTHALMOLOGICAL SECTION

PRESIDENT: Roy O. Schulz, M.D.
SECRETARY: Arnall Patz, M.D., 1212 Eutaw Place, Baltimore

BALTIMORE EAR, NOSE AND THROAT SOCIETY

PRESIDENT: Robert Z. Berry, M.D.
SECRETARY: Francis Catlin, M.D., John Hopkins Hospital, Baltimore

Massachusetts

MASSACHUSETTS MEDICAL SOCIETY
SECTION ON OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Virgil Casten, M.D.
SECRETARY: Robert E. Klotz, M.D., 243 Charles St., Boston

BOSTON CITY HOSPITAL AURAL AND OPHTHALMIC ALUMNI SOCIETY

PRESIDENT: D. Robert Alpert, M.D.
SECRETARY: Frederick L. Landrigan, M.D., 520 Commonwealth Ave., Boston

Michigan

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON OPHTHALMOLOGY

CHAIRMAN: Paul L. Cusick, M.D.
SECRETARY: Paul Van Portfliet, M.D., 26 Sheldon Avenue S.E., Grand Rapids

MICHIGAN STATE MEDICAL SOCIETY, SECTION ON OTOLARYNGOLOGY

CHAIRMAN: V. E. Cortopassi, M.D.
SECRETARY: Donald S. Bolstad, M.D., Henry Ford Hospital, Detroit
TIME AND PLACE: September 28, 1961; Pantland Hotel, Grand Rapids, Mich.

MICHIGAN TRIOLOGICAL SOCIETY

PRESIDENT: John E. Magielski, M.D.
SECRETARY: Walter Z. Rundles, Jr., M.D., 500 South Grand Traverse, Flint 3
TIME AND PLACE: Five times yearly; Lansing or Marshall

DETROIT OPHTHALMOLOGICAL CLUB

PRESIDENT: Members in rotation
SECRETARY-TREASURER: Horace L. Weston, M.D., 703 Mutual Bldg., Detroit 26
TIME AND PLACE: Second Tuesday of November, December, February, and March; Wayne County Medical Society Bldg., Detroit

DETROIT OPHTHALMOLOGICAL SOCIETY

PRESIDENT: James A. Olson, M.D.
SECRETARY: Lester E. McCullough, M.D., 1711 David Whitney Bldg., Detroit 26
TIME AND PLACE: November 10; December 8; January 26; February 23, and March 23; Wayne County Medical Society Bldg.

DETROIT OTOLARYNGOLOGICAL SOCIETY

PRESIDENT: Ned I. Chalat, M.D.
SECRETARY: Harold Schuknecht, M.D., Henry Ford Hospital, Detroit
TIME AND PLACE: 7:00 p.m., Third Wednesday of each month; Wayne County Medical Society Bldg., Detroit

SAGINAW VALLEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Joseph H. Bergin, M.D.
SECRETARY: Arno Weiss, M.D., 3521 State St., Saginaw
TIME AND PLACE: First Tuesday of each month; High-Life Inn, Saginaw

Minnesota

MINNESOTA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Paul G. Bunker, M.D.
SECRETARY-TREASURER: Richard O. Leavenworth, Jr., M.D., 4959 Excelsior Blvd., Minneapolis 16
TIME AND PLACE: Second Friday of each month; Minneapolis Club, Minneapolis; Minnesota Club, St. Paul

Missouri

KANSAS CITY SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Gunnar O. Proud, M.D.
SECRETARY: Earl G. Padfield, Jr., M.D., 4706 Broadway, Kansas City, Mo.

EAR, NOSE AND THROAT CLUB OF ST. LOUIS

CHAIRMAN: Alfred J. Cone, M.D.
SECRETARY: Daniel D. Klaff, M.D., 3720 Washington Blvd., St. Louis 8
TIME AND PLACE: Third Wednesday of January, March, May and November; University Club

ST. LOUIS OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Philip T. Shahan, M.D.
SECRETARY: Robert A. Moses, M.D., 640 South Kingshighway, St. Louis 10
TIME AND PLACE: 6:30 p.m., 4th Thursday each month, except November; Park Plaza Hotel

Montana

MONTANA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Cecil Hall, M.D.
SECRETARY: James Morrison, M.D., 1241 N. Broadway, Billings

Nebraska

NEBRASKA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Paul Peterson, M.D.
SECRETARY: E. S. Maness, M.D., 1006 Sharp Bldg., Lincoln
TIME AND PLACE: April, 1961; Omaha

OMAHA EYE , EAR, NOSE AND THROAT SOCIETY

PRESIDENT: L. T. Placek, M.D.
SECRETARY: W. C. McIntire, M.D., 3610 Dodge Street, Omaha
TIME AND PLACE: Third Wednesday of January, March, May, September, and November; Omaha Club, 20th and Douglas, Omaha

New Jersey

ACADEMY OF MEDICINE OF NEW JERSEY EYE, EAR, NOSE AND THROAT SECTION

CHAIRMAN: Herbert L. Moskowitz, M.D.
SECRETARY: Leon Schwartz, M.D., 162 Chestnut Street, Rutherford
TIME AND PLACE: Third Friday of each month; Newark Eye and Ear Infirmary

MEDICAL SOCIETY OF NEW JERSEY, SECTION ON OPHTHALMOLOGY

PRESIDENT: Louis A. Amdur, M.D.
SECRETARY: Henry Abrams, M.D., 253 Witherspoon Street, Princeton
TIME AND PLACE: March, 1961; Haddon Hall, Atlantic City

NEW JERSEY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: E. A. Atwood, M.D.
SECRETARY: John Scillieri, M.D., 660 Broadway, Paterson
TIME AND PLACE: May, 1961; Atlantic City

NEW JERSEY OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Samuel Diskan, M.D.
SECRETARY: Oram Kline, Jr., M.D., 524 Cooper Street, Camden
TIME AND PLACE: November, Princeton; May, Atlantic City

New Mexico

NEW MEXICO OPHTHALMOLOGICAL SOCIETY

PRESIDENT: J. A. Dillahunt, M.D.
SECRETARY: James T. McGuckin, M.D., 107 Girard Blvd. S. E., Albuquerque

New York

NEW YORK ACADEMY OF MEDICINE, SECTION ON OPHTHALMOLOGY

CHAIRMAN: Joseph Laval, M.D.
SECRETARY: Joseph A. C. Wadsworth, M.D., 108 East 68th Street, New York 21
TIME AND PLACE: Third Monday of each month; Afternoon sessions at hospitals (2:30 p.m.), evening sessions at New York Academy, room 20 (8:00 p.m.)

NEW YORK ACADEMY OF MEDICINE, SECTION ON OTOLARYNGOLOGY

CHAIRMAN: Darrell G. Voorhees, M.D.
SECRETARY: Duncan R. McCuaig, M.D., 81 East 77th Street, New York City
TIME AND PLACE: 8:30 p.m., Third Wednesday of each month; New York Academy of Medicine, 2 E. 103rd St., New York 29

NEW YORK SOCIETY FOR CLINICAL OPHTHALMOLOGY

PRESIDENT: Alfred Kestenbaum, M.D.
SECRETARY: Alan H. Barnert, M.D., 960 Park Avenue, New York 28
TIME AND PLACE: First Monday of the month, November thru May; New York Academy of Medicine, 2 E. 103rd St., New York 29

CENTRAL NEW YORK EYE, EAR, NOSE AND THROAT SOCIETY

PRESIDENT: George Sisson, M.D.
SECRETARY: Wesley Bradley, M.D., 1100 E. Genessee St., Syracuse

EASTERN NEW YORK EYE, EAR, NOSE AND THROAT ASSOCIATION

PRESIDENT: W. R. Updegraff, M.D.
SECRETARY: Frank C. Furlong, M.D., 713 Union St., Schenectady
TIME AND PLACE: First Thursday of November, January, February, March, April and June; Albany, Schenectady and Troy

ALUMNI ASSOCIATION OF THE NEW YORK EYE AND EAR INFIRMARY

PRESIDENT: Joseph H. Krug, M.D.
SECRETARY: John R. Finlay, M.D., Greenwich Medical Bldg., 49 Lake Ave., Greenwich, Conn.

SECTION ON LARYNGOLOGY, RHINOLOGY AND OTOLOGY OF THE MEDICAL SOCIETY OF THE COUNTY OF KINGS AND ACADEMY OF MEDICINE OF BROOKLYN

PRESIDENT: Nathan Thaler, M.D.
SECRETARY-TREASURER: Adolph Wolferman, M.D., 153 Pierrepont Street, Brooklyn 1

BROOKLYN OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Ira W. Mensher, M.D.
SECRETARY: Nicholas P. Tantillo, M.D., One Hanson Place, Brooklyn 17
TIME: Fourth Monday of November, January, March and May

BUFFALO OPHTHALMOLOGICAL CLUB

PRESIDENT: Charles H. Addington, M.D.
SECRETARY: John P. Luhr, M.D., 40 North Street, Buffalo 2
TIME AND PLACE: Second Thursday, October thru May; Sheraton Hotel, Buffalo

LONG ISLAND OPHTHALMOLOGICAL SOCIETY

PRESIDENT: John R. Roche, M.D.
SECRETARY: Gerald M. Branower, M.D., 36 Barstow Road, Great Neck, N. Y.
TIME AND PLACE: Dinner Meetings; Fourth Monday of February, April, September and November

North Carolina

NORTH CAROLINA EYE, EAR, NOSE AND THROAT SOCIETY

PRESIDENT: George T. Noel, M.D.
SECRETARY: John S. Gordon, M.D., 1350 South Kings Drive, Charlotte, N. C.

Ohio

AKRON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: C. T. Korsmo, M.D.
SECRETARY-TREASURER: F. M. Krichbaum, M.D., 175 South Main Street, Akron 8
TIME AND PLACE: 6:30 p.m., First Monday of October, December, February and May; Akron City Club, Akron

CINCINNATI OTOLARYNGOLOGICAL SOCIETY

PRESIDENT: Raymond L. Pfister, M.D.
SECRETARY: William Schrimpf, M.D., 3035 Clinton Avenue, Cincinnati 20
TIME AND PLACE: Third Monday of each month; Academy of Medicine Bldg., Cincinnati

CLEVELAND OPHTHALMOLOGICAL CLUB

PRESIDENT: Russell J. Nicholl, M.D.
SECRETARY: John H. Graham, M.D., 5370 Pearl Road, Parma 29

CLEVELAND OTOLARYNGOLOGICAL CLUB

PRESIDENT: R. W. Machamer, M.D.
SECRETARY: Fred Tingwald, M.D., 2060 East 93rd Street, Cleveland
TIME AND PLACE: Second Wednesday of November, February, April and May; Wade Park Manor

COLUMBUS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Morris L. Battles, M.D.
SECRETARY-TREASURER: Richard H. Wehr, M.D., 150 Broad Street, Columbus 15
TIME AND PLACE: First Monday of each month, October through April; University Club, 40 S. Third St., Columbus

TOLEDO EYE, EAR, NOSE AND THROAT SOCIETY

PRESIDENT: Gail Ann Locken, M.D.
SECRETARY: Jerry W. Draheim, M.D., 1838 Parkwood
TIME AND PLACE: 6:00 p.m., Third Tuesday of each month, November through June; Toledo Club

Oklahoma

OKLAHOMA CITY ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Jack Hough, M.D.
SECRETARY: J. C. Pickard, M.D., Pasteur Medical Bldg., Oklahoma City

Oregon

OREGON ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: George Saunders, M.D.
SECRETARY: Donald C. Mettler, M.D., 1216 S. W. Yamhill St., Portland
TIME AND PLACE: Fourth Tuesday of each month; Aero Club of Oregon, 804 S.W. Taylor St., Portland

Pennsylvania

PENNSYLVANIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: John Knox Covey, M.D.
SECRETARY: Daniel S. De Stio, M.D., 121 South Highland Ave., Pittsburgh
TIME AND PLACE: May 18-20, 1961; Bedford Springs Hotel, Bedford, Pa.

WESTERN PENNSYLVANIA EYE, EAR, NOSE AND THROAT SOCIETY

PRESIDENT: Edwin C. Miller, M.D.
SECRETARY-TREASURER: F. E. Murdock, M.D., 28½ W. Scribner Ave., DuBois
TIME AND PLACE: Third Thursday of May and October; Indiana, Pennsylvania

WYOMING VALLEY EYE, EAR, NOSE AND THROAT CLUB

PRESIDENT: W. C. Marsden, M.D.
SECRETARY: Albert J. Abbott, M.D., 203 E. Green St., Nanticoke
TIME AND PLACE: Last Tuesday of each month, October through April; Luzerne County Medical Society Bldg., S. Franklin St., Wilkes-Barre

PHILADELPHIA COLLEGE OF PHYSICIANS, SECTION ON OPHTHALMOLOGY

SECRETARY: Thomas H. Cowan, M.D., 1930 Chestnut Street, Philadelphia
TIME AND PLACE: Third Thursday of each month, October to May; College of Physicians, 19 S. 22nd St., Philadelphia

PHILADELPHIA COUNTY MEDICAL SOCIETY, EYE SECTION

CHAIRMAN: William C. Frayer, M.D.
SECRETARY: Ralph C. Lanciano, M.D., 259 South 17th Street, Philadelphia
TIME AND PLACE: Third Thursday of each month; Philadelphia County Medical Society Bldg., 301 S. 21st St., Philadelphia

PHILADELPHIA LARYNGOLOGICAL SOCIETY

PRESIDENT: Joseph P. Atkins, M.D.
SECRETARY: Charles M. Norris, M.D., 3401 North Broad Street, Philadelphia 40
TIME AND PLACE: First Tuesday of October, December, April and May; College of Physicians Bldg., Philadelphia

PITTSBURGH OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Harvey E. Thorpe, M.D.
SECRETARY: Myles M. Berk, M.D., 4101 Jenkins Arcade, Pittsburgh 22
TIME AND PLACE: Third Monday of each month; Mellon Institute

PITTSBURGH OTOLOGICAL SOCIETY

PRESIDENT: Carson S. Dimling, M.D.
SECRETARY: Clyde B. Lamp, Jr., M.D., 8102 Jenkins Arcade, Pittsburgh 22

READING EYE, EAR, NOSE AND THROAT SOCIETY

PRESIDENT: Robert Shoemaker, M.D.
SECRETARY: James H. Parker, Jr., M.D., 238 N. Fifth St., Reading
TIME AND PLACE: 5:30 p.m., Third Wednesday of each month, September to June; Wyomissing Club

South Carolina

SOUTH CAROLINA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: S. H. Ross, M.D.
SECRETARY-TREASURER: Roderick Macdonald, M.D., 330 E. Main St., Rock Hill
TIME AND PLACE: September 11 and 12, 1961; Frances Marion Hotel, Charleston, S. C.

Tennessee

TENNESSEE STATE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Ralph O. Rychener, M.D.
SECRETARY: William F. Murrah, Jr., M.D., 130 Madison Avenue, Memphis
TIME AND PLACE: April 10 and 11, 1961; Chattanooga, Tenn.

CHATTANOOGA AND HAMILTON COUNTY EYE, EAR, NOSE AND THROAT SOCIETY

CHAIRMAN AND SECRETARY: J. E. Johnson, M.D., 4547 Brainerd Rd., Chattanooga 4
TIME AND PLACE: 6:30 p.m., Last Thursday of each month; Mountain City Club

KNOXVILLE SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: E. E. Miller, M.D.
SECRETARY: Reese Patterson, M.D., 406 West Church, Knoxville
TIME AND PLACE: Four times yearly; University of Tennessee Research Hospital

MEMPHIS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

CHAIRMAN: Each member in alphabetical order
SECRETARY: William F. Murrah, M.D., 1720 Exchange Bldg., Memphis
TIME AND PLACE: Second Tuesday of each month; Memphis Eye, Ear, Nose and Throat Hospital

NASHVILLE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Phillip L. Lyle, M.D.
SECRETARY: Clyde Alley, M.D., Medical Arts Bldg., Nashville
TIME AND PLACE: Third Monday, October through May; Caduceus Club, Mid State Medical Center

Texas

TEXAS SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: D. Gatlin Mitchell, M.D.
SECRETARY: Oliver W. Suehs, M.D., 14 Medical Arts Square, Austin, Texas
TIME AND PLACE: December 2 and 3, 1961; Sheraton Hotel, Dallas

DALLAS ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: James L. Baldwin, M.D.
SECRETARY-TREASURER: Marvin G. Shepard, M.D., 722 Medical Arts Bldg.
TIME AND PLACE: First Tuesday each month, Parkland Hospital

FORT WORTH EYE, EAR, NOSE AND THROAT SOCIETY

PRESIDENT: Jack Brownfield, M.D.
SECRETARY: Joe L. Bussey, M.D., 1201 W. Presidio, Fort Worth
TIME AND PLACE: First Friday of each month; St. Joseph Hospital

HOUSTON OPHTHALMOLOGICAL SOCIETY

PRESIDENT: Richard E. Leigh, Jr., M.D.
SECRETARY: Carey J. Hargrove, M.D., 1216 Memorial Professional Bldg.
TIME AND PLACE: 8:00 p.m.; First Thursday, September through June; Doctor's Club

SAN ANTONIO SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Graham B. Milburn, M.D.
SECRETARY: Thomas F. Hogan, Jr., M.D.
CORRESPONDING SECRETARY: J. L. Mims, Jr., M.D., 705 E. Houston St., San Antonio
TIME: Third Tuesday of each month; September through May

TEXAS OPHTHALMOLOGICAL ASSOCIATION

PRESIDENT: Otto Lippmann, M.D.
SECRETARY: James H. Scruggs, M.D., 2223 Austin Avenue, Waco
TIME AND PLACE: April 24 and 25, 1961; Galveston

Utah

UTAH OTO-OPHTHALMOLOGICAL SOCIETY

PRESIDENT: David Dolowitz, M.D.
SECRETARY: Don B. McAffee, M.D., 508 East South Temple, Salt Lake City
TIME AND PLACE: Third Monday of each month; University Club, Salt Lake City

Virginia

VIRGINIA SOCIETY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: R. O. Smith, M.D.
SECRETARY-TREASURER: Marion K. Humphries, Jr., M.D., 104 E. Market St., Charlottesville
TIME AND PLACE: May 4-6, 1961; Roanoke

TIDEWATER OPHTHALMOLOGICAL AND OTOLARYNGOLOGICAL SOCIETY

PRESIDENT: Milton Salasky, M.D.
SECRETARY: J. A. Klotz, Jr., M.D., 905 Wainwright Bldg., Norfolk 10
TIME AND PLACE: October, February and May; Tidewater area

RICHMOND, VIRGINIA, EYE, EAR, NOSE AND THROAT SOCIETY

PRESIDENT: John Gill, M.D.
SECRETARY: Mason Smith, M.D., 2035 Monument Ave., Richmond 20
TIME AND PLACE: Third Tuesday of January, March, May and October; Commonwealth Club

Washington

WASHINGTON STATE MEDICAL ASSOCIATION,
SECTION ON EYE, EAR, NOSE AND THROAT

PRESIDENT: Earl L. Barrett, M.D.
SECRETARY: Neil F. Thorlakson, M.D., 605 Cobb Bldg., Seattle 1
TIME AND PLACE: September 19, 1961; Seattle

PUGET SOUND ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: John Shiach, M.D.
SECRETARY: Louis N. Hungerford, Jr., M.D., 1118 9th Avenue, Seattle 1
TIME AND PLACE: Third Tuesday of the month; October through May, Roosevelt Hotel, Seattle

SPOKANE ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: Wilmes Malcolm, M.D.
SECRETARY: Robert J. Davis, South 427 Bernard, Spokane
TIME AND PLACE: Quarterly, Last Tuesday of the month; Medical Library

West Virginia

WEST VIRGINIA ACADEMY OF OPHTHALMOLOGY AND OTOLARYNGOLOGY

PRESIDENT: John A. B. Holt, M.D.
SECRETARY-TREASURER: Worthy W. McKinney, M.D., Beckley, W. Va.
TIME AND PLACE: April 6-8, 1961; Greenbrier Hotel, White Sulphur Springs

Wisconsin

MADISON, WISCONSIN, EYE, EAR, NOSE AND THROAT SOCIETY

PRESIDENT: John V. Berger, Jr., M.D.
SECRETARY: John Doolittle, M.D., 110 East Main Street, Madison
TIME AND PLACE: 6:00 p.m., First Tuesday of each month, November thru May; Madison Club

MILWAUKEE OTO-OPHTHALMIC SOCIETY

PRESIDENT: Roger Lehman, M.D.
SECRETARY: Charles J. Finn, M.D., 161 West Wisconsin Ave., Milwaukee
TIME AND PLACE: Fourth Tuesday of each month, October through May; University Club

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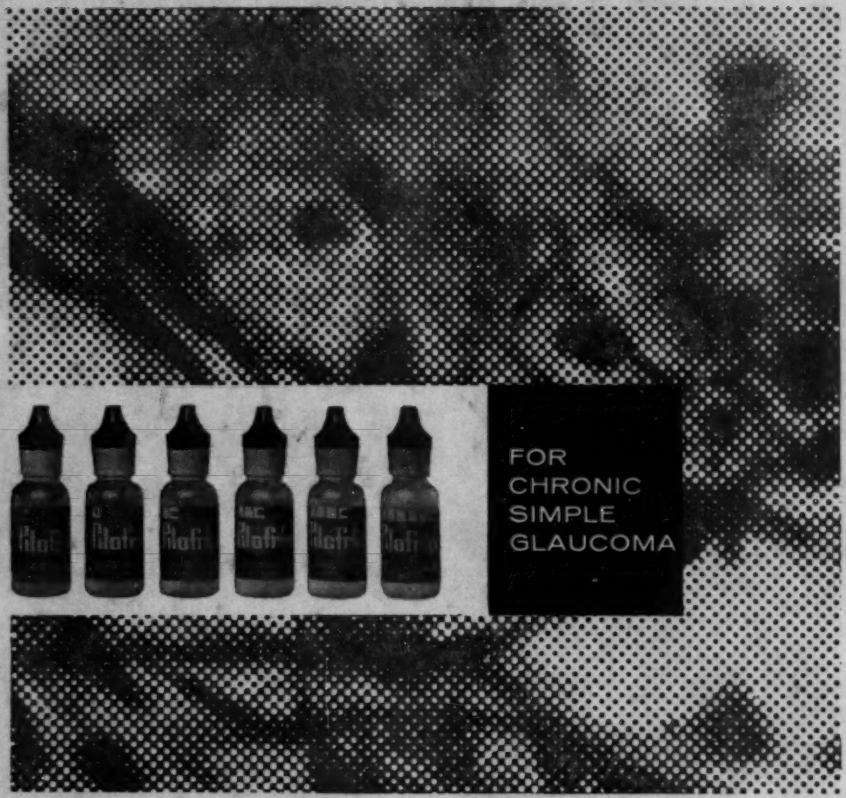

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